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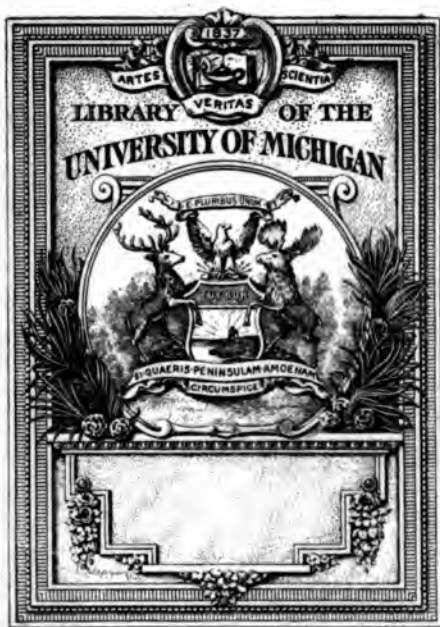
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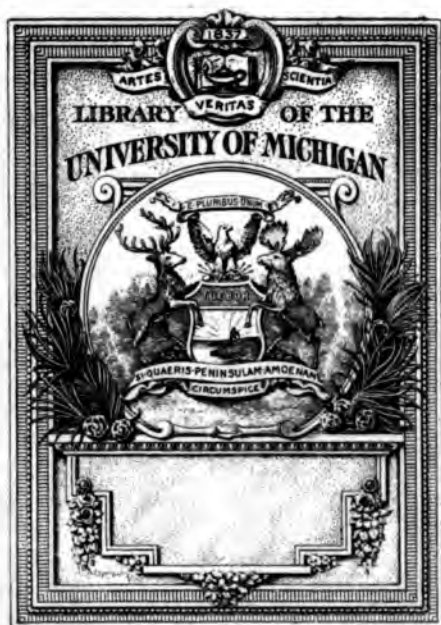
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# MEDICO-CHIRURGICAL TRANSACTIONS.

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1867.



# ROYAL MEDICAL AND CHIRURGICAL SOCIETY OF LONDON.

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FELLOWS OF THE SOCIETY APPOINTED BY  
THE COUNCIL AS REFEREES OF PAPERS,

FOR THE SESSION OF 1867-68.

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BALLARD, EDWARD, M.D.  
BARNES, ROBERT, M.D.  
BASHAM, WILLIAM RICHARD, M.D.  
BURROWS, GEORGE, M.D., F.R.S.  
CLARK, FREDERICK LE GROS.  
COCK, EDWARD.  
CRITCHETT, GEORGE.  
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HANCOCK, HENRY.  
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HOLDEN, LUTHER.  
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JOHNSON, GEORGE, M.D.  
LEE, HENRY.  
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MARSHALL, JOHN, F.R.S.  
MERRIMAN, SAMUEL WILLIAM JOHN, M.D.  
MURCHISON, CHARLES, M.D., F.R.S.  
OGLE, JOHN WILLIAM, M.D.  
OLDHAM, HENRY, M.D.  
POLLOCK, GEORGE DAVID.  
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REES, GEORGE OWEN, M.D., F.R.S.  
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SANDERSON, JOHN BURDON, M.D., F.R.S.  
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SIBSON, FRANCIS, M.D., F.R.S.  
SIEVEKING, EDWARD HENRY, M.D.  
SIMON, JOHN, F.R.S.  
SMITH, SPENCER.  
WEST, CHARLES, M.D.



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FROM ITS FORMATION.

ELECTED

- 1805. WILLIAM SAUNDERS, M.D.
- 1808. MATTHEW BAILLIE, M.D.
- 1810. SIR HENRY HALFORD, BART., M.D., G.C.H.
- 1813. SIR GILBERT BLANE, BART., M.D.
- 1815. HENRY CLINE.
- 1817. WILLIAM BABINGTON, M.D.
- 1819. SIR ASTLEY PASTON COOPER, BART., K.C.H., D.C.L.
- 1821. JOHN COOKE, M.D.
- 1823. JOHN ABERNETHY.
- 1825. GEORGE BIRKBECK, M.D.
- 1827. BENJAMIN TRAVERS.
- 1829. PETER MARK ROGET, M.D.
- 1831. WILLIAM LAWRENCE.
- 1833. JOHN ELLIOTSON, M.D.
- 1835. HENRY EARLE.
- 1837. RICHARD BRIGHT, M.D., D.C.L.
- 1839. SIR BENJAMIN COLLINS BRODIE, BART., D.C.L.
- 1841. ROBERT WILLIAMS, M.D.
- 1843. EDWARD STANLEY.
- 1845. WILLIAM FREDERICK CHAMBERS, M.D., K.C.H.
- 1847. JAMES MONCRIEFF ARNOTT.
- 1849. THOMAS ADDISON, M.D.
- 1851. JOSEPH HODGSON.
- 1853. JAMES COPLAND, M.D.
- 1855. CÆSAR HENRY HAWKINS.
- 1857. SIR CHARLES LOCOCK, BART., M.D.
- 1859. FREDERIC CARPENTER SKEY.
- 1861. BENJAMIN GUY BABINGTON, M.D.
- 1863. RICHARD PARTRIDGE.
- 1865. JAMES ALDERSON, M.D.
- 1867. SAMUEL SOLLY.

# FELLOWS

OF THE

## ROYAL MEDICAL AND CHIRURGICAL SOCIETY

OF LONDON.

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### EXPLANATION OF THE ABBREVIATIONS.

P.—President.	V.P.—Vice-President.
T.—Treasurer.	S.—Secretary.
L.—Librarian.	C.—Member of Council.

The figures succeeding the words *Trans.* and *Pro.* show the number of Papers which have been contributed to the Transactions or Proceedings by the Fellow to whose name they are annexed. *Sci. Com.* is attached to the names of those who have served on the Scientific Committees of the Society.

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**OCTOBER, 1867.**

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Amongst the non-residents, those marked thus (\*) are entitled by composition to receive the Transactions.

#### *Elected*

- 1841 \*ABERCROMBIE, JAMES, M.D., Cape of Good Hope.
- 1846 \*ABERCROMBIE, JOHN, M.D., Physician to the Cheltenham General Hospital, 13, Suffolk square, Cheltenham.
- 1851 \*ACLAND, HENRY WENTWORTH, M.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; Physician to the Radcliffe Infirmary; Regius Professor of Medicine, and Clinical Professor in the University of Oxford.
- 1847 ACOSTA, ELISHA, M.D., 24, Rue du Luxembourg, St. Honoré, Paris.

*Elected*

- 1842 ACTON, WILLIAM, 17, Queen Anne street, Cavendish square.  
*Trans.* 1.
- 1851 ADAMS, JOHN, Surgeon to, and Lecturer on Descriptive and Surgical Anatomy at, the London Hospital; 10, Finsbury Circus. *Trans.* 3.
- 1852 ADAMS, WILLIAM, Surgeon to the Royal Orthopædic Hospital; 5, Henrietta street, Cavendish square.  
*Trans.* 2.
- 1867 AIKIN, CHARLES ARTHUR, 7, Clifton place, Hyde Park.
- 1837 \*AINSWORTH, RALPH FAWSETT, M.D., Consulting Physician to the Manchester Royal Infirmary; Cliff Point, Lower Broughton, Manchester.
- 1839 ALCOCK, SIR RUTHERFORD, K.C.B., K.C.T., K.T.S., D.C.L., H.M.'s Envoy Extraordinary at the Court of Pekin.  
• *Trans.* 1.
- 1826 ALDERSON, JAMES, M.D., F.R.S., President of the Royal College of Physicians; 17, Berkeley square. S. 1829. C. 1848. T. 1849. V.P. 1852-3. P. 1865-6.  
*Trans.* 3.
- 1843 ALDIS, CHARLES JAMES BERRIDGE, M.D., Medical Officer of Health for St. George's, Hanover square; Senior Physician to the Surrey Dispensary; and Physician to the St. Paul and St. Barnabas Dispensary; 1, Chester terrace, Chester square. *Trans.* 2.
- 1850 ALEXANDER, CHARLES REVANS, Surgeon to the Royal Infirmary for Diseases of the Eye; 6, Cork street, Bond street.
- 1866 ALLBUTT, THOMAS CLIFFORD, M.A. and B.M. Camb., F.L.S., Lecturer on the Practice of Physic at the Leeds School of Medicine, and Physician to the Leeds General Infirmary; 38, Park square, Leeds. *Trans.* 2.
- 1863 ALTHAUS, JULIUS, M.D., 18, Bryanston street, Portman square.
- 1862 ANDREW, EDWYN, M.D., Windsor House, Shrewsbury.
- 1862 ANDREW, JAMES, M.D., Assistant Physician to St. Bartholomew's Hospital; 59, Russell square.
- 1820 ANDREWS, THOMAS, M.D., Norfolk, Virginia.

*Elected*

- 1867 ANSTIE, FRANCIS EDMUND, M.D., Senior Assistant Physician and Lecturer on Materia Medica at the Westminster Hospital; 16, Wimpole street.
- 1819 †ARNOTT, JAMES MONCRIEFF, F.R.S., Chapel House, Lady Bank, Fifeshire. L. 1826-8. V.P. 1832-3. T. 1835-40. C. 1846, 1855-6. P. 1847-8. *Trans.* 8.
- 1817 †ASHBURNER, JOHN, M.D., F.L.S., 1, Hyde park place, Cumberland gate. C. 1821, 1830-1.
- 1851 ASHTON, THOMAS JOHN, Consulting Surgeon to the St. Marylebone Infirmary; 31, Cavendish square.
- 1820 \*BADLEY, JOHN, Dudley, Worcestershire.
- 1840 BAINBRIDGE, WILLIAM.
- 1836 BAIRD, ANDREW WOOD, M.D., Physician to the Dover Hospital; 7, Camden crescent, Dover, Kent.
- 1851 \*BAKER, ALFRED, Surgeon to the Birmingham General Hospital, and Lecturer on Surgery at Sydenham College; 20A, Temple row, Birmingham.
- 1865 BAKER, WILLIAM MORRANT, Demonstrator of Anatomy and Operative Surgery, and Warden of the College, St. Bartholomew's Hospital. *Trans.* 2.
- 1839 †BALFOUR, THOMAS GRAHAM, M.D., F.R.S., Deputy Inspector-General of Hospitals; 6, Whitehall yard. C. 1852-3. V.P. 1860-1. *Trans.* 2.
- 1848 BALLARD, EDWARD, M.D., Medical Officer of Health for Islington; 7, Compton terrace, Upper street, Islington. *Trans.* 3.
- 1849 BALLARD, THOMAS, M.D., 10, Southwick place, Hyde park.
- 1866 \*BANKS, JOHN THOMAS, M.D., King's Professor of Physic, Physician to Richmond, Whitworth, and Hardwicke Hospitals, and Sir Patrick Dun's Hospital; Consulting Physician to the Coombe Hospital; 10, Merrion square east, Dublin.
- 1847 BARCLAY, ANDREW WHYTE, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital; Medical Officer of Health for Chelsea; 23A, Bruton street, Berkeley square. S. 1857-60. L. 1861-2. C. 1865-6. *Trans.* 2.

*Elected*

- 1848 BARKER, EDGAR, 9, Oxford square, Hyde park.
- 1862 BARKER, EDGAR, jun., 6, Upper Hyde park street.
- 1833 †BARKER, THOMAS ALFRED, M.D., Senior Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 27, Wimpole street. C. 1844-5. V.P. 1853-4. T. 1860-2. *Trans.* 6.
- 1861 BARNES, ROBERT, M.D., Obstetric Physician to, and Lecturer on Midwifery at, St. Thomas's Hospital, and Physician to the Royal Maternity Charity; 46, Finsbury square. *Trans.* 3.
- 1864 BARRATT, JOSEPH GILLMAN, M.D.; 8, Cleveland gardens, Bayswater.
- 1840 BARROW, BENJAMIN, Surgeon to the Royal Isle of Wight Infirmary; Clifton House, Ryde, Isle of Wight.
- 1859 BARWELL, RICHARD, Assistant-Surgeon to, and Lecturer on Surgical Anatomy at, the Charing Cross Hospital; 32, George street, Hanover square. *Trans.* 1.
- 1844 BASHAM, WILLIAM RICHARD, M.D., Senior Physician to, and Lecturer on Medicine at, the Westminster Hospital; 17, Chester street, Belgrave square. S. 1852-4. C. 1860-1. V.P. 1864-5. *Trans.* 2.
- 1862 BEALE, LIONEL SMITH, M.B., F.R.S., Professor of Physiology and General and Morbid Anatomy in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. *Trans.* 1.
- 1860 BEALEY, ADAM, M.D., M.A. Camb., Physician to the Royal General Dispensary, St. Pancras; 27, Tavistock square.
- 1841 BEAMAN, GEORGE, M.D., 3, Henrietta street, Covent garden.
- 1856 BEARDSLEY, AMOS, Bay villa grange, Newton in Cartmel, Lancashire.
- 1865 BEATTIE, HENRY, M.D., 15, Percy circus, Pentonville.
- 1836 BEAUMONT, WILLIAM RAWLINGS, Consulting Surgeon to the Toronto General Hospital, late Professor of Surgery in the University of King's College; Toronto, Canada West. *Trans.* 3.

*Elected*

- 1840 BEEVOR, CHARLES, 129, Harley street.
- 1858 BEGLEY, WILLIAM CHAPMAN, M.D., Middlesex County Lunatic Asylum, Hanwell.
- 1819 †BELL, THOMAS, F.R.S., F.L.S., The Wakes, Selborne, Hants. C. 1832-3. V.P. 1854. *Trans.* 1.
- 1847 BENNET, JAMES HENRY, M.D., The Ferns, Weybridge, and Mentone.
- 1845 BERRY, EDWARD UNWIN, 76, Gower street, Bedford square.
- 1820 BERTIN, STEPHEN, Paris.
- 1865 \*BICKERSTETH, EDWARD ROBERT, Surgeon to the Royal Infirmary, Liverpool, and Lecturer on Clinical Surgery in the Liverpool School of Medicine; 2, Rodney street, Liverpool.
- 1815 †BILLING, ARCHIBALD, M.D., F.R.S., Member of the Senate of the University of London; 6, Grosvenor gate. C. 1825. V.P. 1828-9.
- 1827 BIRCH, WILLIAM, Barton-under-Needwood, Staffordshire. *Trans.* 2.
- 1855 BIRD, PETER HINCKES, F.L.S., 1, Norfolk square, Hyde park.
- 1856 BIRD, WILLIAM, Surgeon to the West London Hospital; 7, George street, Hanover Square.
- 1849 BIRKETT, EDMUND LLOYD, M.D., Physician to the City of London Hospital for Diseases of the Chest; 48, Russell square. C. 1865-6
- 1851 BIRKETT, JOHN, F.L.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 59, Green street, Grosvenor square. L. 1856-7. S. 1863-5. C. 1867. *Trans.* 5. *Sci. Com.*
- 1846 BIRT, HUGH, 16, Ulster place, Regent's park (abroad).
- 1866 BISHOP, EDWARD, M.D., late Lecturer on Materia Medica and Therapeutics at the Leeds School of Medicine; Cintra park, Upper Norwood.
- 1843 BLACK, PATRICK, M.D., *Vice-President*, Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 11, Queen Anne street, Cavendish square. C. 1856. V.P. 1866.

*Elected*

- 1847 BLACKMAN, GEORGE C., M.D., Professor of Surgery in the Medical College of Ohio; New York, U.S.
- 1840 BLAKISTON, PEYTON, M.D., F.R.S., St. Leonard's-on-Sea.
- 1865 BLANCHET, HILARION, Examiner to the College of Physicians and Surgeons, Lower Canada; 6, Palace street, Quebec, Canada East.
- 1865 BLANDFORD, GEORGE FIELDING, M.B., 3, Clarges street, Piccadilly.
- 1867 BLOXAM, JOHN ASTLEY, Regent's park barracks, Albany street.
- 1823 BOJANUS, LOUIS HENRY, M.D., Wilna.
- 1846 BOSTOCK, JOHN ASHTON, Hon. Surgeon to H.M. the Queen; Surgeon-Major Scots Fusilier Guards; 54, Chester square, Belgravia. C. 1861-2. *Sci. Com.*
- 1863 BOWEN, FRANCIS, M.D., 62, Upper Berkeley street, Portman square.
- 1841 BOWMAN, WILLIAM, F.R.S., Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 5, Clifford street, Bond street. C. 1852-3. V.P. 1862. *Trans.* 3.
- 1862 BRACE, WILLIAM HENRY, Surgeon to the Bath United Hospital; 1, Gay street, Bath.
- 1867 \*BRETT, ALFRED T., M.D., Lime-tree House, Watford, Herts.
- 1851 BRODHURST, BERNARD EDWARD, Assistant-Surgeon to St. George's Hospital, and to the Royal Orthopædic Hospital; 20, Grosvenor street. *Trans.* 2; *Pro.* 1.
- 1844 †BROOKE, CHARLES, M.A., F.R.S., *Librarian*, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; 16, Fitzroy square. C. 1855. L. 1866-67.
- 1854 \*BROWN, HENRY, Windsor, Berks.
- 1857 \*BROWN, ROBERT, Surgeon to the Cumberland Dispensary; 5, Devonshire street, Carlisle.
- 1860 BROWN-SEQUARD, CHARLES EDOUARD, M.D., F.R.S., Professor of Physiology and Pathology, Massachusetts Medical College, Harvard University, Boston, U.S.; Paris. *Sci. Com.*
- 1851 BROWNE, ALEXANDER, M.D., Twynholm, Kirkcudbright.

*Elected*

- 1867 BRUCE, ALEXANDER, Assistant-Curator of the Anatomical and Pathological Museum, University College; 8, Old Cavendish street, Cavendish square.
- 1860 BRYANT, THOMAS, Assistant-Surgeon to, and Demonstrator of Operative Surgery at, Guy's Hospital; 2, Finsbury square. *Trans.* 6; *Pro.* 1. *Sci. Com.*
- 1855 BRYANT, WALTER JOHN, L.R.C.P. Edinb.; 23A, Sussex square, Hyde park gardens.
- 1823 BUCHANAN, B. BARTLET, M.D.
- 1864 BUCHANAN, GEORGE, M.D., Physician to the London Fever Hospital, and Assistant-Physician to the Hospital for Sick Children; Medical Inspector for the Privy Council; Medical Officer of Health for St. Giles District; 53, Harley street, Cavendish square.
- 1864 BUCKLE, FLEETWOOD, M.D.
- 1839 BUDD, GEORGE, M.D., F.R.S., Consulting Physician to the Seamen's Hospital Ship, 'Dreadnought;' Barnstaple, Devon. C. 1846-7. V.P. 1857. *Trans.* 5.
- 1833 †BURROWS, GEORGE, M.D., F.R.S., President of the Medical Council; Consulting Physician to St. Bartholomew's Hospital; Physician to Christ's Hospital; Member of the Senate of the University of London; 18, Cavendish square. C. 1839-40, 1858-9. T. 1845-7. V.P. 1849-50. *Trans.* 2.
- 1820 BURROWS, SAMUEL.
- 1837 BUSK, GEORGE, F.R.S., F.L.S., Surgeon to the Seamen's Hospital Ship 'Dreadnought;' 32, Harley street, Cavendish square. C. 1847-8. V.P. 1855. T. 1866. *Trans.* 4.
- 1818 BUTTER, JOHN, M.D., F.R.S., F.L.S., Physician Extraordinary to the Plymouth Royal Eye Infirmary; Windsor villa, Plymouth.
- 1851 \*CADGE, WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 24, St. Giles's street, Norwich. *Trans.* 1.
- 1851 CALLAWAY, THOMAS, Maison Limozin, Place Bresson, Algiers.



*Elected*

- 1861 CALLENDER, GEORGE WILLIAM, Assistant-Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; 47, Queen Anne street, Cavendish square. *Trans.* 1. *Sci. Com.*
- 1852 \*CANNEY, GEORGE, M.D., Bishop-Auckland, Darlington, Durham.
- 1847 CARLILL, JOHN BURFORD, M.D., Surgeon-Accoucheur to the Newman street Lying-in Institution; 57, Berners st.
- 1853 CARTER, ROBERT BRUDENELL, Stroud, Gloucestershire.
- 1845 CARTWRIGHT, SAMUEL, Professor of Dental Surgery at King's College, London; Surgeon-Dentist to King's College Hospital; 32, Old Burlington street. C. 1860-1. *Sci. Com.*
- 1839 CATHROW, WILLIAM, 42, Weymouth street, Portland place. C. 1860-1.
- 1845 CHALK, WILLIAM OLIVER, Surgeon to the St. Marylebone Eye and Ear Institution; 3, Nottingham terrace, York gate, Regent's park.
- 1844 CHAMBERS, THOMAS KING, M.D., *Vice-President*, Hon. Physician to H.R.H. the Prince of Wales; Consulting Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Consulting Physician to the Lock Hospital; 22B, Brook street, Grosvenor square. *Trans.* 1. C. 1861. V.P. 1867.
- 1859 CHANCE, FRANK, M.D., Croft Lodge, Cambridge.
- 1849 CHAPMAN, FREDERICK, Old Friars, Richmond green, Surrey.
- 1837 CHAPMAN, HENRY THOMAS, 16, Lower Seymour street, Portman square. C. 1858.
- 1852 CHILDS, GEORGE BORLASE, Surgeon-in-Chief to the City Police Force; 11, Finsbury place south.
- 1865 CHOLMELEY, WILLIAM, M.D., Physician to the Great Northern Hospital; 40, Russell square.
- 1842 CHOWNE, WILLIAM DINGLE, M.D., Physician to, and Lecturer on Medicine and Midwifery at, the Charing Cross Hospital; Corresponding Fellow of the Royal Academy of Surgery of Madrid; 17, Hyde park place, Cumberland gate. C. 1853-4.

*Elected*

- 1866 CHURCH, WILLIAM SELBY, M.D., Reader in Anatomy, Christ Church, Oxford; Assistant-Physician to, and Demonstrator of Morbid Anatomy at, St. Bartholomew's Hospital; 41, Bryanston street, Portman square.
- 1860 CLARK, ANDREW, M.D., Physician to, and Lecturer on Medicine at, the London Hospital; 23, Montague place, Russell square.
- 1839 †CLARK, FREDERICK LE GROS, Professor of Human Anatomy and Surgery to the Royal College of Surgeons; Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; Surgeon to the Magdalen Hospital, and Consulting Surgeon to the Royal Surrey County Hospital; Examiner in Surgery to the University of London; 14, St. Thomas's street, Southwark, and Lee, Kent. S. 1847-9. V.P., 1855-6. *Trans.* 3.
- 1848 CLARKE, JOHN, M.D., Obstetric Physician to, and Lecturer on Midwifery at, St. George's Hospital; Physician to the General Lying-in Hospital; 42, Hertford street, May fair. C. 1866.
- 1866 CLARKE, WILLIAM FAIRLIE, Surgeon to the St. George's and St. James's Dispensary; 1, Curzon street, May fair.
- 1861 \*CLARKE, WILLIAM JAMES, Surgeon to the Huddersfield Infirmary; John-William street, Huddersfield, Yorkshire.
- 1850 CLARKSON, JOSIAH, New Hall street, Birmingham. *Trans.* 1.
- 1842 CLAYTON, OSCAR MOORE PASSEY, 5, Harley street. C. 1865.
- 1853 CLOVER, JOSEPH THOMAS, 3, Cavendish place, Cavendish square.
- 1857 COATES, CHARLES, F.R.C.P., Edinb., Physician to the Bath United General Hospital; 10, Circus, Bath.
- 1851 COCK, EDWARD, Senior Surgeon to, and Lecturer on Clinical Surgery at, Guy's Hospital; Consulting Surgeon to the Asylum for Deaf and Dumb; Dean street south, Tooley street, Southwark. C. 1857. *Trans.* 3.

*Elected*

- 1850 COHEN, DANIEL WHITAKER, M.D.; 26, Oxford road, Kilburn.
- 1835 \*COLBORNE, WILLIAM, Chippenham, Wiltshire.
- 1818 COLE, ROBERT, F.L.S., Holybourne, Hampshire.
- 1855 COLLINS, FREDERICK, M.D., Medical Officer of Health for Wanstead; Wanstead lodge, Essex.
- 1867 COOKE, T. C. WEEDEN, Senior Surgeon to the Royal Free Hospital, and to the Cancer Hospital; 76, Upper Berkeley street, Portman square, W.
- 1840 \*COOKE, WILLIAM ROBERT, 2, Carlton villas, Hencroft street, Slough.
- 1865 COOPER, ALFRED, Assistant-Surgeon to St. Mark's Hospital; 70, Jermyn street, Piccadilly.
- 1819 COOPER, GEORGE, Brentford, Middlesex.
- 1841 COOPER, GEORGE LEWIS, one of the Surgeons to the National Vaccine Institution, and Teacher of Vaccination to the Medical School of University College; Surgeon to the Bloomsbury Dispensary; 7, Woburn place, Russell square. C. 1860-1. *Trans.* 1.
- 1843 COOPER, WILLIAM WHITE, Surgeon-Oculist in Ordinary to H.M. the Queen; and Hon. Consulting Ophthalmic Surgeon to St. Mary's Hospital; 19, Berkeley square. C. 1858-9.
- 1841 COOTE, HOLMES, Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; 7, Princes street, Hanover square. S. 1853-4. C. 1864-5. *Trans.* 2.
- 1835 COPELAND, GEORGE FORD, Cheltenham.
- 1822 †COPLAND, JAMES, M.D., F.R.S., Consulting Physician to the Royal Infirmary for Children, and to the Great Northern Hospital; Hon. Fellow of the Royal Academy of Sciences of Sweden, &c.; 5, Old Burlington street, C. 1831. V.P. 1838-9. P. 1853-4.
- 1851 CORBET, JOHN. *Trans.* 2.
- 1860 \*CORRY, THOMAS CHARLES STEUART, M.D., Surgeon to the Belfast General Dispensary; 9, Clarendon place, Belfast.
- 1839 \*CORSELLIS, CHARLES CÆSAR, M.D., F.L.S., Benson, Oxon.

*Elected*

- 1853 CORY, WILLIAM GILLET, M.D.
- 1847 COTTON, RICHARD PAYNE, M.D., Physician to the Hospital for Consumption and Diseases of the Chest; 46, Clarges street, Piccadilly. C. 1863.
- 1828 †COULSON, WILLIAM, Hon. Consulting Surgeon to St. Mary's Hospital, and to the German Hospital; 2, Frederick's place, Old Jewry, and 1, Chester terrace, Regent's park. C. 1831. L. 1832-7. V.P. 1851-2. *Trans.* 1.
- 1864 COULSON, WALTER JOHN, Surgeon to the Lock Hospital, 29, St. James's place.
- 1860 †COUPER, JOHN, Assistant-Surgeon to the London Hospital, and the Royal London Ophthalmic Hospital; 28, Park street, Grosvenor square.
- 1862 COWELL, GEORGE, Surgeon to the St. George's and St. James's Dispensary; 65, Belgrave road, Pimlico.
- 1841 CRAWFORD, MERVYN ARCHDALL NOTT, M.D., Wiesbaden. C. 1853-4.
- 1847 CRITCHETT, GEORGE, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 29, Harley street, Cavendish square. C. 1865. *Trans.* 1.
- 1862 CROMPTON, SAMUEL, 17A, Princess street, Manchester.
- 1837 CROOKES, JOHN FARBAR, 5, Waterloo crescent, Dover.
- 1860 CROSS, RICHARD, M.D., Queen street, Scarborough.
- 1849 \*CROWFOOT, WILLIAM EDWARD, Beccles, Suffolk.
- 1851 CUMMING, JAMES CAMERON, M.D., 1, Cadogan place, Sloane street.
- 1865 CURGENVEN, J. BRENDON, 11, Craven hill gardens, Bayswater.
- 1846 CURLING, HENRY, Surgeon to the Margate Royal Sea-Bathing Infirmary, and the Ramsgate Seamen's Infirmary; Ramsgate, Kent.
- 1837 CURLING, THOMAS BLIZARD, F.R.S., Surgeon to the London Hospital; 39, Grosvenor street. S. 1845-6. C. 1850. T. 1854-7. V.P. 1859. *Trans.* 13. *Pro.* 1. *Sci. Com.*
- 1847 CURREY, JOHN EDMUND, M.D., Lismore, County Waterford.
- 1822 CUSACK, CHRISTOPHER JOHN, Chateau d'Eu, France.

*Elected*

- 1852 CUTLER, THOMAS, M.D., Acting Physician at the Spa Waters; Spa, Belgium.
- 1836 \*DANIEL, JAMES STOCK, Ramsgate, Kent.
- 1848 DAUBENT, HENRY, San Remo, Italy.
- 1846 DAVIES, FREDERICK, M.D., 124, Gower street, Bedford sq.
- 1847 DAVIES, JOHN, M.D., Physician Extraordinary to the Hertford General Infirmary, and Visiting Physician to the Hadham Palace Lunatic Asylum, Hertford.
- 1853 DAVIES, ROBERT COKEB NASH, Rye, Sussex.
- 1852 DAVIES, WILLIAM, M.D., 10, Gay street, Bath.
- 1852 DAVIS, JOHN HALL, M.D., Physician Accoucheur to, and Lecturer on Midwifery at, the Middlesex Hospital; Physician to the Royal Maternity Charity, and Consulting Physician-Accoucheur to the St. Pancras Infirmary; 24, Harley street, Cavendish square.
- 1818 DAWSON, JAMES, Wray Castle, Windermere.
- 1847 DAY, GEORGE EDWARD, M.D., F.R.S., Emeritus Professor of Medicine in the University of St. Andrew's; Furze well House, Torquay.
- 1867 DAY, WILLIAM HENRY, M.D., 10, Manchester square.
- 1858 DELIMA, TROFILO, M.D., Caracas, Venezuela, South America.
- 1867 DE MERIC, VICTOR, Surgeon to the Royal Free Hospital, and to the German Hospital, Dalston; 17, Brook street, Grosvenor square.
- 1846 \*DENTON, SAMUEL BEST, M.D., Ivy Lodge, Hornsea, Hull.
- 1859 DICKINSON, WILLIAM HOWSHIP, M.D., Assistant-Physician to, and Lecturer on Materia Medica at, St. George's Hospital; Assistant-Physician to the Hospital for Sick Children; 11, Chesterfield street, May fair. *Trans. 7. Sci. Com.*
- 1844 DICKSON, ROBERT, M.D., F.L.S., Physician to the Scottish Hospital; 16, Hertford street, May fair. C. 1860.
- 1839 †DIXON, JAMES, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; and Consulting Ophthalmic Surgeon to the Asylum for Idiots: 2, Portman square. L. 1849-55. V.P., 1857-8. T. 1863-4. C. 1866-67. *Trans. 4.*

*Elected*

- 1862 DOBELL, HORACE B., M.D., Physician to the Royal Infirmary for Diseases of the Chest, City road ; 84, Harley street. *Trans.* 1.
- 1845 DODD, JOHN.
- 1857 DOUGLAS, ARCHIBALD, M.D., 8, Clifton place, Sussex square, Hyde park.
- 1863 DOWN, JOHN LANGDON HAYDON, M.D., Assistant-Physician to, and Lecturer on Materia Medica and Therapeutics at, the London Hospital ; Physician to the Asylum for Idiots, Earlswood, Redhill. *Trans.* 2.
- 1867 DRAGE, CHARLES, M.D., Hatfield, Herts.
- 1853 DRUITT, ROBERT, M.R.C.P., Medical Officer of Health for St. George's, Hanover square ; 37, Hertford street, May fair. *Trans.* 2.
- 1865 DRYSDALE, CHARLES ROBERT, M.D., Physician to the Farringdon Dispensary ; Assistant-Physician to the Metropolitan Free Hospital ; 99, Southampton row, Russell square.
- 1865 DUCKWORTH, DYCE, M.D., Medical Tutor to St. Bartholomew's Hospital ; Physician to the Royal General Dispensary ; 70, Wimpole street.
- 1845 DUFF, GEORGE, M.D., High street, Elgin.
- 1845 DUFFIN, EDWARD WILLSON, 18, Devonshire street, Portland place. *Trans.* 1.
- 1867 DUKES, M. CHARLES, M.D., High House, Enfield Highway.
- 1833 †DUNN, ROBERT, 31, Norfolk street, Strand. C. 1845-6. *Trans.* 2.
- 1861 DU PASQUIER, CLAUDIUS FRANCIS, Surgeon-Apothecary to H.M. the Queen, and to the Household of H.R.H. the Prince of Wales ; 62, Pall Mall.
- 1863 DURHAM, ARTHUR EDWARD, Assistant-Surgeon to, and Lecturer on Anatomy at, Guy's Hospital ; 30, Brook street, Grosvenor square. *Trans.* 2. *Sci. Com.*
- 1843 DURRANT, CHRISTOPHER MERCER, M.D., Physician to the East Suffolk and Ipswich Hospital ; Ipswich, Suffolk.
- 1839 DYER, HENRY SUMNER, M.D., 37, Bryanston square. C. 1854-5.
- 1836 EARLE, JAMES WILLIAM, late of Norwich.

*Elected*

- 1824 EDWARDS, GEORGE.
- 1823 EGERTON, CHARLES CHANDLER, Kendall Lodge, Epping.
- 1861 \*ELLIOT, ROBERT, M.D., Physician to the Carlisle Dispensary ; 35, Lowther street, Carlisle.
- 1848 ELLIS, GEORGE VINEY, Examiner in Anatomy at the University of London ; Professor of Anatomy in University College, London. C. 1863-4. *Trans.* 2.
- 1854 \*ELLISON, JAMES, M.D., Surgeon in Ordinary to the Royal Household, Windsor ; 14, High street, Windsor.
- 1835 ENGLAND, WILLIAM, M.D., Ipswich, Suffolk.
- 1842 ERICHSEN, JOHN, Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital ; Examiner in Surgery at the University of London ; 6, Cavendish place, Cavendish square. C., 1855-6. *Trans.* 2.
- 1836 EVANS, GEORGE FABIAN, M.D., Physician to the General Hospital, Birmingham.
- 1815 \*EVANS, GRIFFITH FRANCIS DORSETT, M.D. ; Trewern, Montgomeryshire. C. 1838.
- 1845 EVANS, WILLIAM JULIAN, M.D., Pinner, Middlesex.
- 1864 FAGGE, CHARLES HILTON, M.D., Assistant-Physician to Guy's Hospital, Physician to the Royal Infirmary for Diseases of Children and Women, Waterloo road ; 12, Union street, Southwark.
- 1858 FALCONER, RANDLE WILBRAHAM, M.D., Physician to the Bath United Hospital ; 22, Bennett street, Bath.
- 1862 FARQUHARSON, ROBERT, M.D., Coldstream Guards' Hospital, Vincent square, Westminster.
- 1844 FARRÉ, ARTHUR, M.D., F.R.S., Physician-Accoucheur to H.R.H. the Princess of Wales and H.R.H. the Princess Louis of Hesse ; 12, Hertford street, May fair. C. 1857. V.P. 1864. *Sci. Com.*
- 1863 FENWICK, SAMUEL, M.D., Assistant-Physician to the City of London Hospital for Diseases of the Chest ; 31, Harley street, Cavendish square. *Trans.* 3.

*Elected*

- 1841 **FERGUSSON, SIR WILLIAM, Bart., F.R.S.**, Sergeant-Surgeon to H.M. the Queen; Professor of Surgery in King's College, London, and Surgeon to King's College Hospital; 16, George street, Hanover square. C. 1849-50. V.P. 1863-4, *Trans.* 4.
- 1852 \***FIELD, ALFRED GEORGE**, Surgeon to St. Mary's Hospital, Brighton; 28, Old Steine, Brighton.
- 1849 **FINCHAM, GEORGE TUPMAN, M.D.**, Physician to, and Lecturer on Clinical Medicine at, the Westminster Hospital; 13, Belgrave Road.
- 1866 **FISH, JOHN CROCKETT, B.A., M.B. Camb.**, 8, Fitzroy square.
- 1836 †**FISHER, SIR JOHN WILLIAM**, 5, Grosvenor gate. C. 1843-4.
- 1860 **FITZGERALD, THOMAS GEORGE**, Staff-Surgeon; 6, Whitehall yard.
- 1866 **FITZPATRICK, THOMAS, M.D., M.A.**, Dublin; 30, Sussex gardens, Hyde park.
- 1842 **FLETCHER, THOMAS BELL ELCOCK, M.D.**, Physician to the Birmingham General Hospital; Waterloo street, Birmingham. *Trans.* 1.
- 1864 \***FOLKER, WILLIAM HENRY**, Surgeon to the North Staffordshire Infirmary; Hanley, Stoke-on-Trent, Staffordshire.
- 1848 **FORBES, JOHN GREGORY**, Surgeon to the Metropolitan Convalescent Institution; 9, Devonport street, Hyde park. *Trans.* 2.
- 1852 †**FORSTER, JOHN COOPER**, Assistant-Surgeon to, and Lecturer on Surgery at, Guy's Hospital; Surgeon to the Royal Infirmary for Children, &c.; 10, St. Thomas's street, Southwark. *Pro.* 1.
- 1865 **FOSTER, BALTHAZAR WALTER, M.D.**, Professor of Anatomy at the Queen's College, Birmingham, and Assistant-Physician to the Queen's Hospital; 4, Old Square, Birmingham.
- 1859 **FOX, EDWARD LONG, M.B.**, Physician to the Bristol Royal Infirmary; 1, Chesterfield place, Clifton.



*Elected*

- 1858 \*FOX, WILSON, M.D., Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 22B, Cavendish Square, *Trans.* 2.
- 1841 FRANZ, JOHN CHRISTOPHER AUGUSTUS, M.D.
- 1843 FRASER, PATRICK, M.D. C. 1866.
- 1836 †FRENCH, JOHN GEORGE, Surgeon to the St. James's Infirmary; 41, Great Marlborough street. C. 1852-3.
- 1849 FRERE, ROBERT TEMPLE, M.A., F.R.C.P., 143, Harley street.
- 1846 FULLER, HENRY WILLIAM, M.D., Physician to St. George's Hospital; 13, Manchester square. C. 1862. S. 1864-5. *Trans.* 2.
- 1864 \*GAIRDNER, WILLIAM TENNANT, M.D., Professor of the Practice of Medicine in the University of Glasgow; Physician to the Glasgow Royal Infirmary; 21, Blythswood square, Glasgow.
- 1865 GANT, FREDERICK JAMES, Surgeon and Pathological Anatomist to the Royal Free Hospital; 16, Connaught square, Hyde Park.
- 1867 GARLAND, EDWARD CHARLES, L.R.C.P. Ed., Yeovil, Somerset.
- 1867 GARLIKE, THOMAS W., Tulse Hill, Brixton.
- 1854 GARROD, ALFRED BARING, M.D., F.R.S., Professor of Materia Medica in King's College, London, and Physician to King's College Hospital; 11, Harley street, Cavendish square. *Trans.* 8.
- 1857 GASCOYEN, GEORGE GREEN, *Secretary*, Surgeon to the Lock Hospital; Assistant-Surgeon to, and Lecturer on Descriptive and Surgical Anatomy at St. Mary's Hospital; 48, Queen Anne street, Cavendish square. S. 1866-67. *Trans.* 2. *Sci. Com.*
- 1851 GASKOIN, GEORGE, 3, Westbourne park.
- 1819 GAULTER, HENRY.
- 1848 GAY, JOHN, Senior Surgeon to the Great Northern Hospital, and Consulting Surgeon to the Asylum for Idiots; 10, Finsbury place south.

*Elected*

- 1866 GEE, SAMUEL JONES, M.D., Assistant-Physician to the Hospital for Sick Children ; 46, Queen Anne street, Cavendish square.
- 1821 \*GEORGE, RICHARD FRANCIS, late Senior Surgeon to the Bath General Hospital ; 20, Marlborough buildings, Bath.
- 1864 GIBB, SIR GEORGE DUNCAN, Bart., M.D., LL.D., Assistant-Physician to, and Lecturer on Forensic Medicine at, the Westminster Hospital ; 1, Bryanston street, Portman square. *Trans.* 1.
- 1858 GODFREY, BENJAMIN, M.D., Carlton House, Enfield, Middlesex.
- 1867 GOODEVE, EDWARD, M.B., late Surgeon-Major, H.M.'s Bengal army ; 40, Park street, Grosvenor square.
- 1851 GOODFELLOW, STEPHEN JENNINGS, M.D., Physician to the Middlesex Hospital ; 5, Savile row, Burlington gardens, C. 1864-5. *Trans.* 2.
- 1818 GORDON, JAMES ALEXANDER, M.D., C.B., F.R.S., Pixholme, near Dorking, Surrey. C. 1828. V.P. 1829. *Trans.* 1.
- 1862 GOULSTON, JOHN G., M.D., 30, Clarence street, Liverpool.
- 1851 GOWLLAND, PETER YEAMES, Surgeon to St. Mark's Hospital ; 34, Finsbury square.
- 1844 GRANTHAM, JOHN, Crayford, Kent.
- 1846 GREAM, GEORGE THOMPSON, M. D., Physician-Accoucheur to H.R.H. the Princess of Wales ; 2, Upper Brook street, Grosvenor square. C. 1863.
- 1843 GREENHALGH, ROBERT, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Bartholomew's Hospital ; and Consulting Physician to the Samaritan Free Hospital for Women and Children, and to the City of London Lying-in Hospital ; 77, Grosvenor street.
- 1860 GREENHOW, EDWARD HEADLAM, M.D., Examiner in Forensic Medicine at the University of London ; Assistant-Physician to, and Lecturer on Public Health and on Medical Jurisprudence at, the Middlesex Hospital ; and Consulting Physician to the Western General Dispensary ; 77, Upper Berkeley street, Portman square. *Trans.* 2.

*Elected*

- 1814 GROVE, JOHN, M.D., Salisbury.
- 1852 GROVE, JOHN, West Hill, Wandsworth, Surrey.
- 1860 GUENEAU DE MUSSY, HENRY, M.D., Physician to the French Hospital, Lisle street, Leicester square; 4, Cavendish place, Cavendish square.
- 1849 GULL, WILLIAM WITHEY, M.D., Member of the Senate of the University of London; 26, Brook street, Grosvenor square. C. 1864. *Trans.* 2.
- 1837 GULLY, JAMES MANBY, M.D.; Great Malvern, Worcestershire.
- 1854 HABERSHON, SAMUEL OSBORNE, M.D., *Secretary*, Physician to, and Lecturer on Materia Medica and Therapeutics at, Guy's Hospital; Examiner in Materia Medica at the University of London; 24, Brook street, Grosvenor square. S. 1867. *Trans.* 3.
- 1849 HAILEY, HAMMETT, Newport Pagnell, Bucks.
- 1848 HALLEY, ALEXANDER, M.D., F.G.S., 16, Harley street, Cavendish square.
- 1819 †HAMMERTON THOMAS, 112, Piccadilly. C. 1829-30.
- 1838 HANCOCK, HENRY, Surgeon to the Charing Cross Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; 76, Harley street, Cavendish square. C. 1851.
- 1849 \*HANSARD, RICHARD JAMES, late Surgeon to the Radcliffe Infirmary, Oxford.
- 1848 \*HARCOURT, GEORGE, M.D., Chertsey, Surrey.
- 1836 HARDING, JOHN FOSSE, Mount Sandford, Southborough, Tunbridge Wells. C. 1858-9.
- 1856 HARE, CHARLES JOHN, M.D., 41, Brook street, Grosvenor square.
- 1857 HARLEY, GEORGE, M.D., F.R.S., Professor of Medical Jurisprudence in University College, London; and Physician to University College Hospital; 25, Harley street, Cavendish square. *Trans.* 1. *Sci. Com.* 2.
- 1864 HARLEY, JOHN, M.D., F.L.S., Assistant-Physician to King's College Hospital, and to the London Fever Hospital; 78, Upper Berkeley street, Portman square. *Trans.* 2.
- 1866 HARPER, PHILIP H., 30, Cambridge street, Hyde park.

*Elected*

- 1859 HARRIS, FRANCIS, M.D., Assistant-Physician to, and Lecturer on Botany at, St. Bartholomew's Hospital; Assistant-Physician to the Hospital for Sick Children; 24, Cavendish square.
- 1841 HARVEY, WILLIAM, Surgeon to the Royal Dispensary for Diseases of the Ear, and to the Freemasons' Female Charity, and Aural Surgeon to the Great Northern Hospital; 2, Soho square. C. 1854.
- 1855 HAVILAND, ALFRED, late Surgeon to the Bridgewater Infirmary; (London.)
- 1828 †HAWKINS, CÆSAR HENRY, F.R.S., Sergeant-Surgeon to H.M. the Queen, and Consulting Surgeon to St. George's Hospital; 26, Grosvenor street. C. 1830-1, 1860. V.P. 1838-9. T. 1841-4. P. 1855-6. *Trans.* 12.
- 1838 †HAWKINS, CHARLES, Inspector of Anatomical Schools in London; 27, Savile row, Burlington gardens. C. 1846-7. S. 1850. V.P. 1858. T. 1861-2. *Trans.* 2.
- 1848 HAWKSLEY, THOMAS, M.D., Physician to the Margaret street Dispensary for Consumption and Diseases of the Chest; 70, Brook street, Hanover square.
- 1860 HAYWARD, HENRY HOWARD, Assistant-Dental Surgeon to the Dental Hospital of London; Dental Surgeon to the Hospital for Consumption, Brompton; 38, Harley street, Cavendish square.
- 1861 HAYWARD, WILLIAM HENRY, Church House, Oldbury, near Birmingham.
- 1848 \*HEALE, JAMES NEWTON, M.D., Physician to the Winchester County Hospital; Winchester, Hants.
- 1865 HEATH, CHRISTOPHER, Assistant-Surgeon to, and Demonstrator of Operative Surgery in, University College Hospital; 9, Cavendish place.
- 1850 HEATON, GEORGE, M.D., Boston, U.S.
- 1829 †HEBERDEN, THOMAS, M.D., 98, Park street, Grosvenor sq.
- 1844 HENNEN, JOHN, M.D. L. 1848-50.
- 1849 HENRIQUES, AMOS, M.D., Hon. Physician to the Spanish Embassy; 67, Upper Berkeley street, Portman square.
- 1821 HERBERSKI, VINCENT, M.D., Professor of Medicine in the University of Wilna.

*Elected*

- 1843 HEWETT, PRESCOTT GARDNER, *Vice-President*, Surgeon-Extraordinary to H.M. the Queen ; Surgeon to St. George's Hospital ; 1, Chesterfield street, May fair. C. 1859. V.P. 1866-67. *Trans.* 7. *Sci. Com.*
- 1855 HEWITT, GRAILY, M.D., Professor of Midwifery in University College, London, and Obstetric Physician to University College Hospital ; Physician to the British Lying-in Hospital ; 36, Berkeley square.
- 1853 HEWLETT, THOMAS, Surgeon to Harrow School ; Harrow, Middlesex. *Trans.* 1.
- 1862 HILL, MATTHEW BERKELEY, M.B., Lond., Assistant-Surgeon to University College Hospital, and Surgeon to the Lock Hospital ; 14, Weymouth street, Portland Place.
- 1867 HILL, SAMUEL, M.D., 22, Mecklenburgh square.
- 1854 HILLIER, THOMAS, M.D., Physician to the Hospital for Sick Children ; Physician to the Skin Department of University College Hospital ; and Medical Officer of Health for St. Pancras ; 32, Queen Anne street, Cavendish square. *Trans.* 2.
- 1842 HILLMAN, WILLIAM AUGUSTUS, Senior Assistant-Surgeon to the Westminster Hospital ; 1, Argyll street, Regent street. C. 1858-9.
- 1841 †HILTON, JOHN, F.R.S., Surgeon Extraordinary to H.M. the Queen ; Surgeon to Guy's Hospital ; Consulting Surgeon to the Royal General Dispensary, St. Pancras ; 10, New Broad street, City. C. 1851. V.P. 1863-4. *Trans.* 3.
- 1859 HIRD, FRANCIS, Assistant-Surgeon to, and Lecturer at, the Charing Cross Hospital ; 13, Old Burlington street.
- 1813 HODGSON, JOSEPH, F.R.S., 60, Westbourne terrace, Hyde park gardens. C. 1817. P. 1851-2. *Trans.* 1.
- 1861 \*HOFFMEISTER, WILLIAM CARTER, M.D., Surgeon to H.M. the Queen in the Isle of Wight ; Cowes, Isle of Wight.
- 1843 HOLDEN, LUTHER, Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital ; Surgeon to the Metropolitan Dispensary ; Surgeon to the Foundling Hospital ; 65, Gower street, Bedford square. C. 1859. L. 1865.

*Elected*

- 1814 †HOLLAND, SIR HENRY, Bart., M.D., D.C.L., LL.D.,  
F.R.S., Physician in Ordinary to H.M. the Queen ; 25,  
Brook street, Grosvenor square. C. 1817, 1833-4.  
V.P. 1826, 1840. *Trans.* 1.
- 1861 HOLMAN, WILLIAM HENRY, M.B. Lond., 68, Adelaide road  
south, Hampstead.
- 1856 HOLMES, TIMOTHY, Assistant-Surgeon to, and Lecturer on  
Surgery at, St. George's Hospital, and Surgeon to the  
Hospital for Sick Children ; Surgeon in Chief to the  
Metropolitan Police Force ; 31, Clarges street, Piccadilly.  
*Trans.* 4. *Sci. Com.*
- 1146 HOLT, BARNARD WIGHT, Senior Surgeon to, and Lecturer  
on Clinical Surgery at, the Westminster Hospital ;  
Medical Officer of Health for Westminster ; 14, Savile  
row, Burlington gardens. C. 1862-3.
- 1846 HOLTHOUSE, CARSTEN, Surgeon to, and Lecturer on Surgery  
at, the Westminster Hospital ; Surgeon to the South  
London Ophthalmic Hospital ; 2, Storey's gate, St.  
James's park. C. 1863.
- 1853 HOOD, WILLIAM CHARLES, M.D., F.L.S., Visiting Physician  
in Lunacy to the Court of Chancery ; Croydon Lodge,  
Surrey. *Trans.* 1.
- 1865 HOWARD, BENJAMIN, M.D., 327, West 23rd street, New  
York.
- 1865 HOWARD, EDWARD, M.D., Redhill, Surrey.
- 1828 \*HOWELL, EDWARD, M.D., Senior Consulting Physician to  
the Swansea Infirmary ; 2, South Hill place, Swansea,  
Glamorganshire.
- 1857 HULKE, JOHN WHITAKER, F.R.S., Assistant-Surgeon to, and  
Lecturer on Physiology and Ophthalmic Surgery at,  
the Middlesex Hospital ; Assistant-Surgeon to the  
Royal London Ophthalmic Hospital, Moorfields ; 10,  
Old Burlington street. *Trans.* 4. *Sci. Com.*
- 1857 HULME, EDWARD CHARLES, Surgeon to the Great Northern  
Hospital ; Surgeon to the Central London Ophthalmic  
Hospital ; 38, Gower street, Bedford square. *Trans.* 1.
- 1844 HUMBY, EDWIN, 83, Hamilton terrace, St. John's wood.  
C. 1866-67.

*Elected*

- 1835 HUMPHREY, GEORGE MURRAY, M.D., F.R.S., Surgeon to Addenbrooke's Hospital, and Professor of Human Anatomy and Physiology in the Cambridge University Medical School; Cambridge. *Trans.* 4.
- 1866 HUNTER, CHARLES, 30, Wilton place, Belgrave square.
- 1849 HUSSEY, EDWARD LAW, Senior Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the County Lunatic Asylum and the Warneford Asylum; 104, St. Aldate's, Oxford. *Trans.* 1.
- 1856 HUTCHINSON, JONATHAN, Surgeon to, and Lecturer on Surgery at, the London Hospital; Assistant-Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 4, Finsbury circus. *Trans.* 1. *Pro.* 2.
- 1820 HUTCHINSON, WILLIAM, M.D.
- 1840 HUTTON, CHARLES, M.D., Senior Physician to the General Lying-in Hospital; 26, Lowndes street, Belgrave square. C. 1858-9.
- 1866 ILES, FRANCIS HENRY WILSON, M.D., Watford, Herts.
- 1847 IMAGE, WILLIAM EDMUND, Senior Surgeon to the Suffolk General Hospital; Bury St. Edmunds, Suffolk. *Trans.* 1.
- 1856 INGLIS, CORNELIUS, M.D., 10, New Cavendish street, Portland place.
- 1841 †JACKSON, PAUL, 24, Wimpole street, Cavendish square. C. 1862.
- 1863 JACKSON, THOMAS VINCENT, Surgeon to the South Staffordshire General Hospital; Darlington street, Wolverhampton.
- 1841 JACBOVICS, MAXIMILIEN MORRIS, M.D., Vienna.
- 1825 JAMES, JOHN B., M.D.
- 1844 JEAFFRESON, SAMUEL JOHN, M.D., Consulting Physician to the Warneford Hospital, and Warwick Dispensary; Leamington, Warwickshire.
- 1839 JEFFREYS, JULIUS, F.R.S., Drymona, Belvidere road south, Upper Norwood, Surrey.
- 1840 \*JENKS, GEORGE SAMUEL, M.D., 18, Circus, Bath.

*Elected*

- 1851 JENNER, WILLIAM, M.D., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Physician to University College Hospital; 18, Harley street, Cavendish square. C. 1864. *Trans.* 3.
- 1848 JOHNSON, ATHOL ARCHIBALD WOOD, 20, Regency square, Brighton. *Trans.* 1.
- 1851 JOHNSON, EDMUND CHARLES, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Genevois."
- 1847 JOHNSON, GEORGE, M.D., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-3. *Trans.* 6.
- 1862 JONES, CHARLES HANDFIELD, M.B., F.R.S., Physician to, and Lecturer on Medicine at, St. Mary's Hospital; 49, Green street, Grosvenor square.
- 1844 †JONES, HENRY BENCKE, M.A., M.D., F.R.S., 31, Brook street, Grosvenor square. C. 1855-6. V.P. 1866. *Trans.* 11.
- 1835 †JONES, HENRY DERVICKE, 12, Norfolk Crescent, Oxford square. C. 1854-5.
- 1837 JONES, THOMAS WILLIAM, M.D., 55, St. John's Park, Upper Holloway, and 3, North buildings, Finsbury.
- 1859 JONES, WILLIAM PRICE, M.D., Surbiton, Kingston.
- 1865 JORDAN, FURNEAUX, Surgeon to the Queen's Hospital, and Senior Surgeon to the Birmingham Eye and Ear Hospital; 16, Colmore row, Birmingham.
- 1816 \*KAUFFMAN, GEORGE HERMANN, M.D., Hanover.
- 1848 \*KENDELL, DANIEL BURTON, M.D., Kettlethorpe Hall, Wakefield, Yorkshire.
- 1847 KEYSER, ALFRED, 21, Norfolk crescent, Oxford square.
- 1857 KJALLMARK, HENRY WALTER, 66, Prince's square, Baywater.
- 1851 KINGDON, JOHN ABERNETHY, Surgeon to the City of London Truss Society, and to the City Dispensary; 2, New Bank buildings, City. C. 1866-7. *Trans.* 1. *Sci. Com.*



*Elected*

- 1855 LANE, JAMES ROBERT, Surgeon to, and Lecturer on Operative Surgery at, St. Mary's Hospital, and Surgeon to the Lock and St. Mark's Hospitals; 2, Berkeley street, Piccadilly. *Trans.* 1.
- 1840 LANE, SAMUEL ARMSTRONG, Surgeon to, and Lecturer on Clinical Surgery at, St. Mary's Hospital, and Consulting Surgeon to the Lock Hospital; 2, Berkeley street, Piccadilly. C. 1849-50. V.P. 1865.
- 1865 LANGTON, JOHN, Assistant-Surgeon to, and Demonstrator of Anatomy at, St. Bartholomew's Hospital; Assistant-Surgeon to the City of London Truss Society; the College, St. Bartholomew's Hospital.
- 1841 \*LASHMAR, CHARLES, M.D., 83, North End, Croydon, Surrey.
- 1862 LATHAM, PETER WALLWORK, M.A., M.B., Physician to Addenbrooke's Hospital, Cambridge; Examiner for Medical Degrees in Cambridge University; 15, Sidney street, Cambridge.
- 1816 LAWRENCE, G. E.
- 1840 LAYCOCK, THOMAS, M.D., F.R.S.E., Professor of the Practice of Medicine and of Clinical Medicine, and Lecturer on Psychology and Mental Diseases in the University of Edinburgh, and Physician to the Edinburgh Royal Infirmary; 4, Rutland street, Edinburgh.
- 1843 \*LEACH, JESSE, Moss Hall, Heywood, near Bury, Lancashire.
- 1822 LEDSAM, JOHN JOSEPH, M.D., 17, Esplanade, Scarborough, Yorkshire.
- 1823 †LEE, HENRY, M.D., Weather Oak, Alvechurch, near Bromsgrove, Worcestershire. C. 1837. S. 1839-40.
- 1843 LEE, HENRY, Surgeon to, and Lecturer on Pathology at, St. George's Hospital; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. *Trans.* 9. *Pro.* 1. *Sci. Com.*
- 1822 †LEE, ROBERT, M.D., F.R.S., Corresponding Member of the Imperial Academy of Medicine, Paris; 4, Savile row, Burlington Gardens. C. 1829, 1834. S. 1830-3. V.P. 1835. *Trans.* 27.

*Elected*

- 1836 LEIGHTON, FREDERICK, M.D., Frankfort-on-the-Maine.
- 1854 LEON, HANANEL DE, M.D., 15, Holland villas road, North Kensington.
- 1806 LIND, JOHN, M.D.
- 1819 LLOYD, ROBERT, M.D.
- 1820 LOCHER, J. G., M.C.D., Town Physician of Zurich. *Trans.* 2.
- 1824 †LOCOCK, SIR CHARLES, Bart., M.D., F.R.S., First Physician Accoucheur to H.M. the Queen; Member of the Senate of the University of London; 26, Hertford street, May fair. C. 1826. V.P. 1841. P. 1857-8. *Trans.* 1.
- 1852 LODGE, CHARLES, M.D., "United States Army."
- 1846 LOMAX, HENRY THOMAS, Surgeon to the County Police; St. Mary's grove, Stafford.
- 1860 LONGMORE, THOMAS, C.B., Deputy Inspector-General, and Professor of Clinical and Military Surgery, New Army Medical School, Royal Victoria Hospital, Netley, Southampton. *Trans.* 2.
- 1836 LÖWENFELD, JOSEPH S., M.D., Berbice.
- 1852 LUKE, JAMES, F.R.S., Consulting Surgeon to the London Hospital, Woolley Lodge, Maidenhead Thicket, Berks. C. 1858. *Trans.* 4.
- 1857 LYON, FELIX WILLIAM, M.D., Littlecote, Hungerford, Hants.
- 1867 MABERLY, GEORGE FREDERICK, Hillside House, Godalming, Surrey.
- 1862 \*M'DONNELL, ROBERT, M.D., F.R.S., Surgeon to Steevens' Hospital; 14, Lower Pembroke street, Dublin. *Trans.* 1.
- 1846 M'EWEN, WILLIAM, M.D., Surgeon to Chester Castle; 27, Nicholas street, Chester.
- 1866 MACGOWAN, ALEXANDER THORBURN, Mooreland House, Martock, Somersetshire.
- 1823 †MACILWAIN, GEORGE, Consulting Surgeon to the Finsbury Dispensary, and the St. Anne's Society's Schools; 3, the Court yard, Albany. C. 1829-30. V.P. 1848. *Trans.* 1.
- 1822 MACINTOSH, RICHARD, M.D.
- 1859 \*M'INTYRE, JOHN, M.D., Odiham, Hants.

*Elected*

- 1818 **MACKENZIE, WILLIAM, M.D.**, Surgeon Oculist to H.M. the Queen in Scotland, and Surgeon to the Glasgow Eye Infirmary; 49, Bath street, Glasgow. *Trans.* 2.
- 1854 \***MACKINDER, DRAPER, M.D.**, Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.
- 1844 **MACLACHLAN, DANIEL, M.D.**, Deputy Inspector-General of Hospitals; Ventnor, Isle of Wight. C. 1860-1. *Trans.* 1.
- 1860 **MACLEAN, JOHN, M.D.**, 24, Portman street, Portman square.
- 1849 **MACLURE, DUNCAN MACLACHLAN, M.B.**, 34, Harley street, Cavendish square.
- 1842 **MACNAUGHT, JOHN, M.D.**, Bedford street, Liverpool.
- 1855 **MARCEY, WILLIAM, M.D.**, F.R.S., 48, Harley street. *Trans.* 2. *Sci. Com.*
- 1848 **MARKHAM, WILLIAM ORLANDO, M.D.**, Physician to St. Mary's Hospital; Poor Law Inspector for the Metropolitan District; 8, Harley street. C. 1862-3. *Trans.* 2.
- 1867 **MARSH, F. HOWARD**, 19A, Golden square.
- 1838 **MARSH, THOMAS PARR, M.D.**
- 1851 **MARSHALL, JOHN, F.R.S.**, Professor of Surgery in University College, London, and Surgeon to University College Hospital; 10, Savile row, Burlington gardens. C. 1866. *Trans.* 2.
- 1841 **MARTIN, SIR JAMES RANALD, C.B.**, F.R.S., Examining Medical Officer to the Secretary of State for India in Council, President of Medical Board for Examination of Officers of H.M.'s Indian Medical Service; Inspector General of Hospitals; 37, Upper Brook street. C. 1853. V.P. 1862.
- 1853 **MASFEN, WILLIAM EDWARD**, Surgeon to the Staffordshire General Infirmary; Stafford.
- 1864 **MASON, FRANCIS**, Assistant-Surgeon to the Westminster Hospital, 10, Conduit street, Regent street.
- 1837 **MAYO, THOMAS, M.D.**, F.R.S., the Refuge, Yarmouth, Isle of Wight. S. 1841. C. 1847-8. V. P., 1851-2.
- 1839 **MEADE, RICHARD HENRY**, Senior Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

*Elected*

- 1865 MEDWIN, AARON GEORGE, M.D., 11, Montpellier row, Blackheath, Kent.
- 1867 MEREDYTH, COLOMIATI, M.D., 76, Margaret street, Cavendish square.
- 1837 MERRIMAN, SAMUEL WILLIAM JOHN, M.D., Consulting Physician-Accoucheur to the Westminster General Dispensary, 13, Godolphin road, New road, Shepherd's Bush. C. 1851-2. *Trans.* 1.
- 1852 MERRYWEATHER, JAMES, Consulting Surgeon to the National Dental Hospital; 57, Brook street, Grosvenor square.
- 1847 MERYON, EDWARD, M.D., F.G.S., 14, Clarges street, Piccadilly. L. 1859-60. C. 1864-5. *Trans.* 2.
- 1815 MEYER, AUGUSTUS, M.D., St. Petersburg.
- 1840 MIDDLEMORE, RICHARD, Consulting Surgeon to the Birmingham Eye Infirmary; Temple row, Birmingham.
- 1854 MIDDLESBROUGH, EDWARD ARCHIBALD, late of Richmond, Surrey.
- 1860 \*MILES, HERBERT CHALMERS, Assistant-Surgeon in the Royal Horse Artillery, Preston Barracks, Brighton; (Naval and Military Club, Cambridge House, Piccadilly.)
- 1818 \*MILLER, PATRICK, M.D., F.R.S.E., Senior Physician to the Devon and Exeter Hospital, and to St. Thomas's Hospital for Lunatics; the Grove, Exeter, Devonshire.
- 1863 MONRO, HENRY, M.D., Physician to St. Luke's Hospital; 13, Cavendish square.
- 1844 MONTEFIORE, NATHANIEL, 36, Hyde park gardens.
- 1848 MOORE, CHARLES HEWITT, *Vice-President*, Surgeon to the Middlesex and St. Luke's Hospitals; 102, Piccadilly. L. 1858. S. 1859-62. C. 1864-5. V.P. 1866-7. *Trans.* 10. *Sci. Com.*
- 1836 MOORE, GEORGE, M.D., Hastings, Sussex.
- 1861 MOREHEAD, CHARLES, M.D., Hon. Surgeon to H.M. the Queen; Deputy-Inspector General of Hospitals; 34, Melville street, Edinburgh.
- 1857 MORGAN, JOHN, 3, Sussex place, Hyde park gardens. *Trans.* 1.

*Elected*

- 1861 MORGAN, JOHN EDWARD, M.B., Lecturer on Pathology at the Manchester Royal School of Medicine; 3, Gore street, Piccadilly, Manchester.
- 1851 MOUAT, FREDERIC JOHN, M.D., Surgeon-Major, Bengal Army; Inspector-General of Gaols in the Lower Provinces of the Bengal Presidency, and Member of the Senate of the University of Calcutta. (The Athenæum Club.)
- 1856 MURCHISON, CHARLES, M.D., F.R.S., Senior Physician to the London Fever Hospital; Physician to, and Lecturer on the Practice of Medicine at, the Middlesex Hospital; 79, Wimpole-street, Cavendish square. *Trans.* 3.
- 1847 MURCHISON, SIMON, Bicester, Oxon.
- 1868 MYERS, ARTHUR B. R., Coldstream Guards' Hospital, Vincent square, Westminster.
- 1859 NAYLER, GEORGE, Assistant-Surgeon to the Hospital for Diseases of the Skin, Blackfriars; Assistant-Surgeon to the Royal Orthopædic Hospital; 8, George street, Hanover square.
- 1835 †NELSON, THOMAS ANDREW, M.D., 10, Nottingham terrace, York gate, Regent's park.
- 1843 NEWTON, EDWARD, 4, Upper Wimpole street. C. 1863-4.
- 1849 NORMAN, HENRY BURFORD, Portland Lodge, Southsea, Hants.
- 1845 NORRIS, HENRY, Charmouth, Dorset.
- 1847 \*NOURSE, WILLIAM EDWARD CHARLES, 11, Marlborough place, Brighton.
- 1849 NOVERRE, ARTHUR, 25, South street, Park lane.
- 1864 NUNN, THOMAS WILLIAM, Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.
- 1859 \*NUNNELEY, THOMAS, Senior Surgeon to the Leeds Eye and Ear Infirmary; Leeds. *Trans.* 3.
- 1847 O'CONNOR, THOMAS, March, Cambridgeshire.
- 1843 O'CONNOR, WILLIAM, M.D., Senior Physician to the Royal Free Hospital; 30, Upper Montagu street, Montagu square.

*Elected*

- 1858 OGLE, JOHN WILLIAM, M.D., Physician to, and Lecturer on Pathology at, St. George's Hospital; 13, Upper Brook street, Grosvenor square. *Trans.* 4.
- 1855 \*OGLE, WILLIAM, M.A., M.D., Physician to the Derby Infirmary; 3, Stewart terrace, Derby.
- 1860 OGLE, WILLIAM, M.D., Lecturer on Physiology at St. George's Hospital, and Physician to the St. George's and St. James's Dispensary; 34, Clarges street, Piccadilly.
- 1850 OLDHAM, HENRY, M.D., Obstetric Physician to, and Clinical Lecturer on Midwifery at, Guy's Hospital; 26, Finsbury square. C. 1865. *Trans.* 1.
- 1846 \*ORMEROD, EDWARD LATHAM, M.D., Physician to the Sussex County Hospital; 14, Old Steine, Brighton. *Trans.* 2.
- 1847 \*PAGE, WILLIAM BOUSFIELD, Surgeon to the Cumberland Infirmary; Carlisle. *Trans.* 2.
- 1840 †PAGET, JAMES, F.R.S., *Treasurer*, Sergeant-Surgeon Extraordinary to H.M. the Queen; Surgeon in Ordinary to H.R.H. the Prince of Wales; Surgeon to, and Lecturer on Surgery at, St. Bartholomew Hospital, and Surgeon to Christ's Hospital; Member of the Senate of the University of London; 1, Harewood place, Hanover square. C. 1848-9. V.P. 1861. T. 1867. *Trans.* 8. *Sci. Com.*
- 1858 \*PALEY, WILLIAM, M.D., Physician to the Ripon Dispensary; Ripon, Yorkshire.
- 1836 PARKER, LANGSTON, Hon. Surgeon to the Queen's Hospital, Birmingham; Colmore row, Birmingham.
- 1847 PARKER, NICHOLAS, M.D., Paris.
- 1841 PARKIN, JOHN, M.D., Rome.
- 1851 PART, JAMES, M.D., 7, Camden road villas (89, Camden road), Camden town.
- 1828 †PARTRIDGE, RICHARD, F.R.S., Professor of Anatomy to the Royal Academy of Arts, Surgeon to King's College Hospital, and Professor of Anatomy in King's College, London; 17, New street, Spring gardens. S. 1832-6. C. 1837-8, 1861-2. V.P. 1847-8. P. 1863-4.

*Elected*

- 1865 PAVY, FREDERICK WILLIAM, M.D., F.R.S., Assistant-Physician to, and Lecturer on Physiology and Comparative Anatomy at, Guy's Hospital; 35, Grosvenor street.
- 1845 PEACOCK, THOMAS BEVILL, M.D., *Vice-President*, Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 20, Finsbury circus. S. 1855-6. V.P. 1867. *Trans.* 2.
- 1864 PEARSON, DAVID RITCHIE, M.D., 23, Upper Phillimore place, Kensington.
- 1856 PEIRCE, RICHARD KING, 16, Norland place, Notting hill.
- 1830 PELECHIN, CHARLES P., M.D., St. Petersburg.
- 1855 \*PEMBERTON, OLIVER, Surgeon to the Birmingham General Hospital, and Lecturer on Surgical Pathology at Sydenham College; 18, Temple row, Birmingham. *Trans.* 1.
- 1844 PETTIGREW, WILLIAM VESALIUS, M.D., Surgeon to the Female Orphan Asylum; 7, Chester street, Grosvenor place.
- 1848 PHILLIPS, EDWARD, M.D., F.L.S., Physician to the Coventry and Warwickshire Hospital; Coventry, Warwickshire.
- 1852 PHILLIPS, RICHARD, 52, Leinster square, Westbourne grove.
- 1854 PHILLIPS, THOMAS BACON, M.D., Physician to the Brighton and Hove Dispensary; 36, Lansdowne place, Brighton.
- 1846 PHILP, FRANCIS RICHARD, M.D., Colby House, Kensington, and Sherborne House, Harrogate, Yorkshire.
- 1867 PICK, THOMAS PICKERING, Demonstrator of Anatomy, and Curator of the Pathological Museum at St. George's Hospital; 9, Bolton row, May fair.
- 1851 \*PICKFORD, JAMES HOLLINS, M.D., M.R.I.A., 1, Cavendish place, Brighton.
- 1836 †PILDUCK, ISAAC, M.D., Physician to the Bloomsbury Dispensary; 22, Montague street, Russell square. *Pro.* 2.
- 1841 PITMAN, HENRY ALFRED, M.D., *Treasurer*, Consulting Physician to St. George's Hospital and to the Royal General Dispensary, St. Pancras; 39, Chester terrace, Regent's park. L. 1851-3. C. 1861-2. T. 1863-7.

*Elected*

- 1850 POLAND, ALFRED, Surgeon to Guy's Hospital, and to the Eye Infirmary attached to the Hospital. (Abroad.) C. 1865-6.
- 1845 POLLOCK, GEORGE DAVID, Surgeon in Ordinary to H.R.H. the Prince of Wales; Surgeon to St. George's Hospital; 27, Grosvenor street. C. 1856-7. L. 1859-62. *Trans.* 2.
- 1865 POLLOCK, JAMES EDWARD, M.D., Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square.
- 1843 POPE, CHARLES, M.D., Glastonbury, Somersetshire.
- 1846 POTTER, JEPHSON, M.D., F.L.S., Physician to the Liverpool General Hospital for Consumption and Diseases of the Chest; 109, Upper Parliament Street, Liverpool.
- 1842 POWELL, JAMES, M.D.
- 1867 POWER, HENRY, Assistant-Surgeon to the Westminster Hospital; 43, Upper Seymour street, Portman sq.
- 1851 POWER, ROBERT FRANCIS, M.D., 71, Gloucester place, Portman square.
- 1857 PRIESTLEY, WILLIAM OVEREND, M.D., Physician-Accoucheur to H.R.H. the Princess Louis of Hesse; Professor of Midwifery in King's College, London; and Physician for the Diseases of Women and Children to King's College Hospital; Examiner in Midwifery at the University of London; Consulting Physician-Accoucheur to the St. Marylebone Infirmary; 17, Hertford street, May fair. *Sci. Com.*
- 1845 PYLE, JOHN, 59, Oxford terrace, Hyde park. C. 1860.
- 1850 QUAIN, RICHARD, M.D., Physician to the Hospital for Consumption and Diseases of the Chest; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. *Trans.* 1. *Sci. Com.*
- 1835 †QUAIN, RICHARD, F.R.S., Surgeon Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery, University College, London; Consulting Surgeon to the Eye Infirmary attached to the Hospital; 32, Cavendish square. C. 1838-9. L. 1846-8. T. 1851-3. V.P. 1856-7. *Trans.* 1. *Pro.* 2.



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- 1852 RADCLIFFE, CHARLES BLAND, M.D., Physician to, and Lecturer on Materia Medica at, the Westminster Hospital; Physician to the National Hospital for the Paralyssed and Epileptic; 25, Cavendish square. C. 1867.
- 1857 RANKE, HENRY, M.D., Munich.
- 1854 RANSOM, WILLIAM HENRY, M.D., Physician to the Nottingham General Hospital; the Pavement, Nottingham.
- 1858 REED, FREDERICK GEORGE, M.D., 46, Hertford street, May fair. *Trans.* 1.
- 1821 REEDER, HENRY, M.D.
- 1857 REES, GEORGE OWEN, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; 26, Albemarle street, Piccadilly. *Trans.* 1.
- 1855 REYNOLDS, JOHN RUSSELL, M.D., Professor of the Principles and Practice of Medicine in University College, London, and Physician to University College Hospital; Physician to the National Hospital for the Paralyssed and Epileptic; 38, Grosvenor street.
- 1865 RHODES, GEORGE WINTER, Surgeon to the Huddersfield Infirmary; 30, Ramsden street, Huddersfield.
- 1847 RICHARDS, SAMUEL, M.D., 36, Bedford square.
- 1852 RICHARDSON, CHRISTOPHER THOMAS, M.B., Warcop, Penrith.
- 1849 \*RICHARDSON, WILLIAM, M.D., 9, Ephraim road, Tunbridge Wells, Kent.
- 1845 RIDGE, BENJAMIN, M.D., 21, Bruton street, Berkeley square.
- 1843 RIDGE, JOSEPH, M.D., 39, Dorset square. C. 1858. *Pro.* 1.
- 1863 RINGER, SYDNEY, M.D., Professor of Materia Medica in University College, London, and Physician to University College Hospital; Assistant-Physician to the Hospital for Sick Children; 15, Cavendish place.
- 1852 ROBERTS, JOHN, M.R.C.P., 56, Grosvenor street.
- 1855 ROBERTSON, CHARLES ALEXANDER LOCKHART, M.D., Medical Superintendent of the Sussex County Lunatic Asylum; Hayward's Heath, Sussex.
- 1857 ROBERTSON, JOHN CHARLES GEORGE, Assistant Medical Officer, Female Department, Middlesex County Lunatic Asylum, Hanwell.

*Elected*

- 1862 ROBINSON, CHARLES, F.R.C.P. Edin., 11, Montagu street, Portman square.
- 1843 ROBINSON, GEORGE, M.D. *Trans.* 2.
- 1843 RODEN, WILLIAM, M.D., the Grange, Kidderminster, Worcestershire.
- 1835 †ROE, GEORGE HAMILTON, M.D., Senior Physician to the Hospital for Consumption and Diseases of the Chest; 124, Park street, Grosvenor sq. C. 1841-2. *Trans.* 1.
- 1836 †ROGERS, ARNOLD, Consulting Surgeon-Dentist to St. Bartholomew's Hospital; 16, Hanover square.
- 1829 ROOTS, WILLIAM SUDLOW, F.L.S., Surgeon to the Royal Establishment at Hampton Court; Kingston, Surrey.
- 1850 ROPER, GEORGE, 180, Shoreditch. (Abroad.)
- 1855 ROSCOW, THOMAS TATTERSALL, M.D.
- 1836 \*ROSE, CALEB BURRELL, F.G.S., 25, King street, Great Yarmouth, Norfolk. *Trans.* 1.
- 1857 ROSE, HENRY COOPER, M.D., Surgeon to the Hampstead Dispensary; High street, Hampstead.
- 1849 ROUTH, CHARLES HENRY FELIX, M.D., Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Trans.* 1.
- 1863 ROWE, THOMAS SMITH, M.D., Surgeon to the Royal Seabathing Infirmary; Cecil street, Margate, Kent.
- 1834 RUMSEY, HENRY WYLDBORE, Wolseley house, Cheltenham.
- 1845 RUSSELL, JAMES, M.D., Physician to the Birmingham General Hospital, and Lecturer on Pathology and Therapeutics at Sydenham College; 91, New Hall street, Birmingham.
- 1851 SALTER, HENRY HYDE, M.D., F.R.S., Physician to, and Lecturer on Medicine at, the Charing Cross Hospital; 14, Harley street, Cavendish square. S. 1866. *Sci. Com.*
- 1856 SALTER, SAMUEL JAMES A., F.R.S., F.L.S., Surgeon-Dentist to, and Lecturer on Dental Surgery at, Guy's Hospital; 17, New Broad street, City. *Trans.* 2.
- 1849 SANDERSON, HUGH JAMES, M.D., Physician to the Hospital for Women; 26, Upper Berkeley street, Portman square.

*Elected*

- 1855 SANDERSON, JOHN BURDON, M.D., F.R.S., Assistant-Physician to, and Lecturer on Physiology at, the Middlesex Hospital; Physician to the Brompton Hospital for Consumption; 49, Queen Anne street, Cavendish square. *Trans. 1. Sci. Com.*
- 1867 SANDFORD, FOLLIOTT JAMES, M.D., Market Drayton, Shropshire.
- 1847 SANKEY, WILLIAM HENRY OCTAVIUS, M.D, Lecturer on Mental Diseases at University College, London; Sandywell park, near Cheltenham.
- 1845 SAUNDERS, EDWIN, Surgeon-Dentist to H.M. the Queen, and to H.R.H. the Prince of Wales; 13A, George street, Hanover square.
- 1834 SAUVAN, LUDWIG V., M.D., Warsaw.
- 1859 SAVORY, WILLIAM SCOVELL, F.R.S., Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Examiner in Physiology and Comparative Anatomy at the University of London; 23A, Brook street, Grosvenor square. *Trans. 3. Sci. Com. 2.*
- 1853 SCHULHOF, MAURICE, M.D., Physician to the Royal General Dispensary, Bartholomew Close; 14, Brook street, Grosvenor square.
- 1861 \*SCOTT, WILLIAM, M.D., Physician to the Huddersfield Infirmary; 12, New North Road, Huddersfield.
- 1858 \*SCRATCHLEY, GEORGE, M.D., New Orleans, Louisiana, U.S. [per Arthur Scratchley, Esq., 8A, Waterloo place, Pall Mall].
- 1863 SEDGWICK, WILLIAM, Surgeon to the St. Marylebone Provident Dispensary; 12, Park place, Upper Baker street.
- 1856 SERCOMBE, EDWIN, Surgeon-Dentist to St. Mary's Hospital; 49, Brook street, Grosvenor square. *Trans. 1. Pro. 1.*
- 1840 SHARP, WILLIAM, M.D., F.R.S., Horton House, Rugby. *Trans. 1.*
- 1837 †SHARPEY, WILLIAM, M.D., F.R.S., LL.D., Professor of Anatomy and Physiology in University College, London; Member of the Senate of the University of London, and Secretary of the Royal Society; University College, and Lawnbank, Hampstead. C. 1848-9. V.P. 1862.

*Elected*

- 1836 †SHAW, ALEXANDER, Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 40, Abbey road west, Kilburn. C. 1842. S. 1843-4. V.P. 1851-2. T. 1858-60. *Trans.* 4.
- 1848 \*SHEARMAN, EDWARD JAMES, M.D., Moorgate, Rotherham, Yorkshire.
- 1859 SIBLEY, SEPTIMUS WILLIAM, Lecturer on Pathological Anatomy at the Middlesex Hospital; 12, New Burlington street. *Trans.* 4. *Sci. Com.*
- 1849 SIBSON, FRANCIS, M.D., F.R.S., Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; Member of the Senate of the University of London; 40, Brook street, Grosvenor square. C. 1863-4. *Trans.* 1. *Sci. Com.*
- 1848 SIEVEKING, EDWARD HENRY, M.D., Physician in Ordinary to H.R.H. the Prince of Wales; Physician to, and Lecturer on Materia Medica at, St. Mary's Hospital; 17, Manchester square. C. 1859-60. S. 1861-3. *Trans.* 2. *Sci. Com.*
- 1842 SIMON, JOHN, F.R.S., Surgeon to, and Lecturer on General Pathology at, St. Thomas's Hospital; Medical Officer of the Privy Council; 8, Richmond terrace, Whitehall, and 40, Kensington square. C. 1854-55. V.P. 1865. *Trans.* 1.
- 1865 SIMS, J. MARION, M.D., Paris.
- 1857 SIORDET, JAMES LEWIS, M.B., Mentone.
- 1824 †SKEY, FREDERIC CARPENTER, F.R.S., Consulting Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; 24, Mount street, Grosvenor square. C. 1828. L. 1829-31. V.P. 1841-2. P. 1859-60. *Trans.* 1.
- 1852 SMITH, CHARLES CASE, Consulting Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk.
- 1866 SMITH, HEYWOOD, M.B., Oxon., Physician-Accoucheur to the St. George's and St. James's Dispensary, 42, Park street, Grosvenor square.
- 1835 SMITH, JOHN GREGORY, Harewood, Leeds, Yorkshire.

*Elected*

- 1843 SMITH, ROBERT WILLIAM, M.D., M.B.I.A., Professor of Surgery in the University of Dublin; Surgeon to the Richmond Hospital; 63, Eccles street, Dublin.
- 1838 SMITH, SPENCER, Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital; 9, Queen Anne street, Cavendish square. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865.
- 1863 SMITH, THOMAS, Assistant-Surgeon to St. Bartholomew's Hospital, and Assistant-Surgeon to the Hospital for Sick Children; 7, Montague street, Russell square. *Trans.* 1. *Sci. Com.*
- 1864 \*SMITH, THOMAS HECKSTALL, Rowlands, St. Mary Cray, Kent.
- 1845 SMITH, WILLIAM, 1, Atlantic terrace west, Weston-super-Mare. *Trans.* 1.
- 1847 SMITH, WILLIAM, M.D., Consulting Physician to the Weymouth Infirmary; Weymouth, Dorsetshire.
- 1850 SMITH, WILLIAM TYLER, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Mary's Hospital; 21, Upper Grosvenor street. C. 1867. *Trans.* 2.
- 1851 SODEN, JOHN, see *Corbet*.
- 1830 †SOLLY, SAMUEL, F.R.S., *President*, Senior Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; Consulting Surgeon to the Royal General Dispensary, Bartholomew Close; 6, Savile row. L. 1838-40. C. 1845-6. V.P. 1849-50. P. 1867. *Trans.* 6.
- 1865 SOUTHAM, GEORGE, Surgeon to the Manchester Royal Infirmary; 21, Lever street, and Oakfield, Pendleton, Manchester. *Trans.* 4.
- 1865 SOUTHEY, REGINALD, M.B., Assistant-Physician and Medical Registrar to St. Bartholomew's Hospital; Physician to the Royal General Dispensary; 32, Montague place, Russell square.
- 1844 SPACKMAN, FREDERICK R., M.D., Harpenden, St. Albans.
- 1834 SPARK, JAMES, Italy.
- 1851 SPITTA, ROBERT JOHN, M.B., Medical Officer to the Clapham General Dispensary; Clapham, Surrey. *Trans.* 1.
- 1843 \*SPRANGER, STEPHEN, Hursley, Hampshire.

*Elected*

- 1867 SQUAREY, CHARLES EDWARD, Resident Medical Officer, University College Hospital. *Trans.* 2.
- 1857 STANTON, JOHN, M.D., 9, Montagu square.
- 1851 STARTIN, JAMES, Surgeon to, and Lecturer on Cutaneous Disorders at, the Hospital for Diseases of the Skin, Blackfriars; 3, Savile row, Burlington gardens.
- 1854 STEVENS, HENRY, M.D., Lond. [79, Grosvenor street].
- 1842 STEWART, ALEXANDER PATRICK, M.D., *Librarian*; 75, Grosvenor street. C. 1856-7. L. 1863-7.
- 1859 STEWART, WILLIAM EDWARD, 12, Weymouth street, Portland place.
- 1856 STOCKER, ALONZO HENRY, M.D., Resident Medical Superintendent of Grove Hall Lunatic Asylum, Bow.
- 1865 STOKES, WILLIAM, Jun., M.D., 5, Merrion square north, Dublin.
- 1843 STORKS, ROBERT REEVE, Paris.
- 1858 †STREATFEILD, JOHN FREMLYN, Assistant-Surgeon to the Royal London Ophthalmic Hospital, Moorfields, and Ophthalmic Surgeon to University College Hospital; 15, Upper Brook street, Grosvenor square.
- 1863 STURGES, OCTAVIUS, M.B., Physician to the Chelsea, Brompton, and Belgrave Dispensary; 85, Wimpole street.
- 1860 SUTRO, SIGISMUND, M.D., Senior Physician to the German Hospital; 37A, Finsbury square.
- 1855 SUTTON, JOHN MAULE, M.D., Bloomfield, Narberth, Pembrokeshire.
- 1861 \*SWEETING, GEORGE BACON, King's Lynn, Norfolk.
- 1842 SYME, JAMES, F.R.S.E., Surgeon in Ordinary to H.M. the Queen in Scotland; Professor of Clinical Surgery in the University of Edinburgh, and Surgeon to the Edinburgh Royal Infirmary; 2, Rutland street, Edinburgh. *Trans.* 5.
- 1854 \*SYMONDS, FREDERICK, Surgeon to the Radcliffe Infirmary and Consulting Surgeon to the Oxford Dispensary; 35, Beaumont street, Oxford.
- 1844 TAMPLIN, RICHARD WILLIAM, Surgeon to the Royal Orthopaedic Hospital; 33, Old Burlington street.

*Elected*

- 1848 TANNER, THOMAS HAWKES, M.D., F.L.S., 9, Henrietta street, Cavendish square.
- 1864 TAUSSIG, GABRIEL, M.D., 70, Piazza Barberini, Rome.
- 1852 TAYLOR, ROBERT, Surgeon to the Central London Ophthalmic Hospital, and to the Cripple's Home, Hill street : 21, Edwards street, Portman square.
- 1845 TAYLOR, THOMAS, Lecturer on Chemistry at the Middlesex Hospital Medical School ; 4, Vere street, Cavendish sq.
- 1856 TRALE, THOMAS PRIDGIN, F.R.S., F.L.S., Surgeon to the Leeds General Infirmary ; Albion st., Leeds. *Trans.* 2.
- 1859 TZGART, EDWARD, 49, Jermyn street, St. James's.
- 1862 THOMPSON, EDMUND SYMES, M.D., Assistant-Physician to King's College Hospital, and to the Hospital for Consumption, Brompton ; 3, Upper George street, Portman square.
- 1857 THOMPSON, HENRY, M.D., Physician to, and Lecturer on Materia Medica at, the Middlesex Hospital ; 52, Welbeck street, Cavendish square.
- 1852 THOMPSON, SIR HENRY, Knt., Surgeon Extraordinary to H.M. the King of the Belgians, Professor of Clinical Surgery in University College, and Surgeon to University College Hospital ; 35, Wimpole street, Cavendish square. *Trans.* 3.
- 1862 THOMPSON, REGINALD EDWARD, M.D., Physician to the St. George's and St. James's Dispensary ; 21, South street, Park lane. *Sci. Com.*
- 1836 THURNAM, JOHN, M.D., Resident Medical Superintendent of the Wilts County Asylum, Devizes, Wiltshire. *Trans.* 4.
- 1848 TILT, EDWARD JOHN, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity ; 60, Grosvenor street.
- 1867 TONGE, MORRIS, M.D., Lecturer on Physiology at the Charing Cross Hospital ; 5, Bolton Row, May Fair.
- 1828 TORRIE, JAMES, M.D.
- 1867 TROTTER, JOHN WILLIAM, Assistant-Surgeon, Coldstream Guards ; Hospital, Vincent square, Westminster.

*Elected*

- 1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household ; 23, Old Burlington street.
- 1864 TUFNELL, THOMAS JOLLIFFE, Examiner in Surgery to the Royal College of Surgeons of Ireland ; 58, Lower Mount street, Merrion square, Dublin.
- 1862 TUKE, THOMAS HARRINGTON, M.D., Manor House, Chiswick, and 37, Albemarle street.
- 1855 TULLOCH, JAMES STEWART, M.D., 1, Pembridge place, Bayswater.
- 1864 TURNER, GEORGE, 9, Sussex gardens, Hyde park.
- 1845 TURNER, THOMAS, F.L.S., Consulting Surgeon to the Manchester Royal Infirmary, and Lecturer on Anatomy and Physiology at the Manchester Royal School of Medicine ; 77, Mosley street, Manchester.
- 1806 VAUX, BOWYER, Teignmouth, Devon.
- 1865 VERNON, BOWATER JOHN, Assistant-Demonstrator of Anatomy, St. Bartholomew's Hospital.
- 1867 VINTRAS, ACHILLE, M.D., Physician to the French Hospital, Lisle street, Leicester square ; 141, Regent street.
- 1828 VULFES, BENEDETTO, M.D., Physician to the Hospital of Aversa, and the Hospital of Incurables, Naples.
- 1854 WADDINGTON, EDWARD, Kettlethorpe Hall, Newark, Notts.
- 1841 WADE, ROBERT, Senior Surgeon to the Westminster General Dispensary ; 68, Dean street, Soho. *Trans.* 1.
- 1864 WAITE, CHARLES DERBY, M.B., 3, Old Burlington street.
- 1867 \*WALLIS, GEORGE, House Surgeon, Addenbrooke's Hospital, Cambridge.
- 1861 \*WALSH, JAMES, M.D., Staff-Surgeon, R.N., 41, Cathrine street, Limerick, Ireland.
- 1852 WALSH, WALTER HAYLE, M.D., Emeritus Professor of the Principles and Practice of Medicine, University College, London ; Consulting Physician to the Hospital for Consumption ; 37, Queen Anne street, Cavendish square. *Trans.* 1.
- 1851 WALTON, HENRY HAYNES, Surgeon to the Central London Ophthalmic Hospital, and Surgeon to St. Mary's Hospital ; 69, Brook street, Hanover square. *Trans.* 1. *Pro.* 1.



*Elected*

- 1852 WANE, DANIEL, M.D., 20, Grafton street, Berkeley square.
- 1821 WARD, WILLIAM TILLZARD.
- 1858 WARDELL, JOHN RICHARD, M.D., 4, Belmont, Tunbridge Wells.
- 1846 WARE, JAMES THOMAS, Consulting Surgeon to the Finsbury Dispensary, and Hon. Surgeon to the Metropolitan Convalescent Institution; 18, Gordon square.
- 1818 WARE, JOHN, Clifton Down, near Bristol.
- 1814 †WARE, MARTIN, 18, Gordon square. C. 1844-5. T. 1846. V.P. 1853.
- 1866 WARING, EDWARD JOHN, M.D., 28, George street, Hanover square.
- 1861 WATERS, A. T. HOUGHTON, M.D., Physician to the Liverpool Northern Hospital, and Lecturer on Anatomy and Physiology in the Liverpool Royal Infirmary School of Medicine; 27, Hope street, Liverpool. *Trans.* 3.
- 1837 WATSON, SIR THOMAS, Bart., M.D., D.C.L., F.R.S.; Physician Extraordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 16, Henrietta street, Cavendish square. C. 1840-1, 1852. V.P. 1845-6.
- 1861 WATSON, WILLIAM SPENCER, M.B., Assistant Surgeon to King's College Hospital; 27, Montague street, Russell square.
- 1854 WEBB, WILLIAM, M.D., Wirksworth, Derbyshire.
- 1840 WEBB, WILLIAM WOODHAM, M.D., Cliff House, Kirtley, South Lowestoft, Suffolk.
- 1842 WEBER, FREDERIC, M.D., 44, Green street, Park lane. C. 1857. V.P. 1865.
- 1857 WEBER, HERMANN, M.D., Physician to the German Hospital; 49, Finsbury square. *Trans.* 5.
- 1835 †WEBSTER, JOHN, M.D., F.R.S., Physician to the Scottish Hospital, and Consulting Physician to the St. George's and St. James's Dispensary. C. 1843-4. V.P. 1855-6. *Trans.* 6. *Pro.* 1.
- 1844 WEGG, WILLIAM, M.D., Physician to the St. George's and St. James's Dispensary; 26, Park lane. L. 1854-8. C. 1861-2.

*Elected*

- 1861 WELLS, JOHN SOELBERG, Professor of Ophthalmology in King's College, London, and Ophthalmic Surgeon to King's College Hospital; Assistant-Surgeon to the Royal London Ophthalmic Hospital; 16, Savile row.
- 1854 WELLS, THOMAS SPENCER, Surgeon in Ordinary to H.M.'s Household; Surgeon to the Samaritan Free Hospital for Women and Children; 3, Upper Grosvenor street. *Trans.* 6. *Pro.* 1.
- 1842 †WEST, CHARLES, M.D., Physician to the Hospital for Sick Children; 61, Wimpole street, Cavendish square. C. 1855-6. V.P. 1863. *Trans.* 2. *Sci. Com.*
- 1828 WHATLEY, JOHN, M.D.
- 1849 WHITE, JOHN.
- 1852 WIBLIN, JOHN, M.D., Medical Inspector of Emigrants and Recruits; Southampton. *Trans.* 1.
- 1844 WILDBORE, FREDERIC, 1 Trafalgar place east, [245] Hackney road.
- 1837 WILKS, GEORGE AUGUSTUS FREDERICK, M.D., [5, Lincoln's Inn Fields.]
- 1863 WILKS, SAMUEL, M.D., Physician to, and Lecturer on Medicine at, Guy's Hospital, Examiner in Medicine at the University of London; 11, St. Thomas's street, Southwark.
- 1865 WILLETT, ALFRED, Assistant-Surgeon to St. Bartholomew's Hospital; 36, Wimpole street, Cavendish square.
- 1864 WILLETT, EDMUND SPARSHALL, M.D., Resident Physician, Wyke House, Isleworth, Middlesex.
- 1860 WILLIAMS, ARTHUR WYNN, M.D., Physician-Accoucheur to the Western General Dispensary; 1, Montagu square.
- 1840 WILLIAMS, CHARLES JAMES BLASIUS, M.D., F.R.S., Consulting Physician to the Hospital for Consumption; 49, Upper Brook street, Grosvenor square. C. 1849-50. V.P. 1860-1. *Sci. Com.*
- 1859 \*WILLIAMS, CHARLES, Surgeon to the Norwich Dispensary; 9, Prince of Wales road, Norwich.
- 1866 WILLIAMS, CHARLES THEODORE, M.B., Oxon., Assistant-Physician to the Hospital for Consumption and Diseases of the Chest at Brompton; 49, Upper Brook street, Grosvenor square.

*Elected*

- 1859 WILLIAMS, JOSEPH, M.D., 8, Tavistock square.
- 1829 WILLIS, ROBERT, M.D., Barnes, Surrey. L. 1838-41.
- 1839 †WILSON, ERASMUS, F.R.S., Consulting Surgeon to the St. Pancras Infirmary; 17, Henrietta street, Cavendish square. *Trans.* 2.
- 1863 WILSON, ROBERT JAMES, L.R.C.P. Edin., 24, Grand Parade, St. Leonards-on-Sea, Sussex.
- 1850 \*WISE, ROBERT STANTON, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Banbury, Oxfordshire.
- 1825 WISE, THOMAS ALEXANDER, M.D., Rostellan Castle, Rostellan, County Cork.
- 1841 WOOD, GEORGE LEIGHTON, Surgeon to the Bath General Hospital; 27, Queen square, Bath.
- 1851 WOOD, JOHN, Assistant-Surgeon to King's College Hospital, and Demonstrator of Anatomy in King's College, London; 4, Montague street, Russell square. C. 1867. *Trans.* 2.
- 1848 WOOD, WILLIAM, M.D., Physician to St. Luke's Hospital for Lunatics; 99, Harley street. C. 1867.
- 1833 †WORMALD, THOMAS, Consulting Surgeon to St. Bartholomew's Hospital; 42, Bedford row. C. 1839. V.P. 1854.
- 1842 WORTHINGTON, WILLIAM COLLINS, Senior Surgeon to the Lowestoft Infirmary; Lowestoft, Suffolk. *Trans.* 3.
- 1865 WOTTON, HENRY, JUN., Cecil House, King's Road, Brighton.
- 1848 WRIGHT, EDWARD JOHN, 13, Montague place, Clapham road.
- 1855 WRIGHT, HENRY G., M.D., Physician to the Samaritan Free Hospital for Women and Children; 66, Harley street, Cavendish square.
- 1860 WYATT, JOHN, Surgeon-Major, Coldstream Guards; Hospital, Vincent square, Westminster.

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[It is particularly requested that any change of Title, Appointment, or Residence, may be communicated to the Secretaries before the 1st of October in each year, in order that the List may be made as correct as possible.]

## HONORARY FELLOWS.

(Limited to Twelve.)

*Elected*

- 1835 BREWSTER, SIR DAVID, K.H., D.C.L., LL.D., F.R.S., Corresp. Memb. Institute of France, Principal and Vice-Chancellor of the University of Edinburgh.
- 1853 BRODIE, SIR BENJAMIN COLLINS, Bart., M.A., F.R.S., Waynflete Professor of Chemistry at Oxford; Cowley House, Oxford.
- 1847 CHADWICK, EDWIN, late Commissioner of the Board of Health.
- 1862 DAUBENY, CHARLES GILES BRIDLE, M.D., LL.D., F.R.S., Hon. M.R.I.A., &c., Professor of Botany, Oxford.
- 1857 FARR, WILLIAM, M.D., D.C.L., F.R.S., General Register Office, Somerset House, and Southlands, Bromley, Kent.
- 1841 HERSCHEL, SIR JOHN FREDERICK WILLIAM, Bart., D.C.L., F.R.S., Corresp. Memb. Institute of France; Collingwood, near Hawkhurst, Kent.
- 1866 MURCHISON, SIR RODERICK IMPEY, Bart., K.C.B., D.C.L. Oxon., LL.D. Camb., M.A., F.R.S., Director-General of the Geological Survey; 16, Belgrave Square.
- 1847 OWEN, RICHARD, D.C.L., LL.D., F.R.S., Corresp. Memb. Institute of France (Foreign Associate of the Academy of Sciences), Superintendent of the Natural History Departments in the British Museum; Sheen Lodge, Mortlake.
- 1825 SEDGWICK, The Rev. ADAM, A.M., D.C.L., LL.D., F.R.S., Woodwardian Professor of Geology, Cambridge.

# FOREIGN HONORARY FELLOWS.

Entered in 1822

## *Electus*

- 1841 ANTEL G. M.D., Member of the Institute and of the Imperial Academy of Medicine, Physician in Ordinary to the Emperor of the French ; Paris.
- 1862 CATTENHETER JEAN M.D., Physician to the "Hôpital de la Charité." Member of the Imperial Academy of Medicine, &c. : Paris.
- 1864 DONDERS FRANK CORNELIUS M.D., Professor of Physiology and Ophthalmology at the University of Utrecht.
- 1856 DUBOIS, BARON PAUL, Commander of the Legion of Honour, Member of the Imperial Academy of Medicine, late Dean of the Faculty of Medicine : Paris.
- 1835 EKSTRÖMER CARL JOHAN, M.D., C.M., K.P.S., and W., Physician to the King of Sweden, President of the College of Health, and Director-General of Hospitals ; Stockholm.
- 1841 EHRENBURG, CHRISTIAN GOTTFRIED, Member of the Institute of France ; Berlin.
- 1866 HANNOVER, ADOLPH, M.D., Professor at Copenhagen.
- 1859 HENLE, J., M.D., Professor of Anatomy at Göttingen.
- 1856 LANGENBECK, BERNHARD, M.D., Professor of Surgery in the University of Berlin.
- 1843 LIEBIG, BARON JUSTUS von, M.D., Foreign Associate of the Academy of Sciences, Conservator of the Royal Collection, and Professor of Chemistry in the University of Munich.
- 1841 LOUIS, P. C. A., M.D., Honorary Physician to the Hotel-Dieu, Member of the Imperial Academy of Medicine ; Paris.
- 1847 MATTEUCCI, CARLO, Professor in the University of Pisa, Member of the Institute of France, Minister of Public Instruction in Italy.

*Elected*

- 1862 **PIROGOFF, NIKOLAUS, M.D.**, Professor of Surgery to the Medico-Chirurgical Academy in St. Petersburg, Director of the Anatomical Institute, Consulting Physician to the Hospitals Obuchow, Peter-Paul, and Maria Magdalena; St. Petersburg.
- 1850 **ROKITANSKY, CARL, M.D.**, Curator of the Imperial Pathological Museum, and Professor of the University of Vienna. Referee for Medical and University Education to the Austrian Ministry.
- 1856 **STROMEYER, LOUIS, M.D.**, Director-General of the Medical Department of the Army of Hanover; Hanover.
- 1856 **VIRCHOW, RUDOLPH, M.D.**, Professor of Pathological Anatomy in the University of Berlin.



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WOODCUTS.

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## ADVERTISEMENT.

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**THE Council of the Royal Medical and Chirurgical Society** deems it proper to state that the Society does not hold itself in any way responsible for the statements, reasonings, or opinions set forth in the various papers, which, on grounds of general merit, are thought worthy of being published in its Transactions.



**Resolutions relative to the publication of the "Proceedings of the Society."**

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**That Abstracts of the papers read will be furnished to the Journals as heretofore.**

A C A S E  
IN WHICH  
O V A R I O T O M Y  
W A S  
TWICE SUCCESSFULLY PERFORMED ON THE  
SAME PATIENT.

BY  
T. SPENCER WELLS, F.R.C.S.,  
SURGEON IN ORDINARY TO HER MAJESTY'S HOUSEHOLD; SURGEON  
TO THE SAMARITAN HOSPITAL.

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Received Oct. 6th.—Read Nov. 13th, 1866.

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IN the forty-sixth volume of the 'Transactions' of this Society a case is published in which I performed ovariectomy, and removed the right ovary of a patient whose left ovary had been removed nine months before by another surgeon. This patient died on the seventh day after the second operation.

In recording that case I stated that Dr. Atlee, of Philadelphia, had successfully performed ovariectomy upon a patient whose opposite ovary had been removed sixteen years before by Dr. Clay, of Manchester. I have since heard that Dr. Frederic Bird has operated for the second time unsuccessfully upon a patient from whom he had removed one ovary fourteen years before. I have not heard of any case in which ovariectomy has been twice successfully performed upon the same patient by the same surgeon, except that which is now submitted to the Society.

I performed the first operation in the Samaritan Hospital on the 15th of February, 1865. The patient was an unmarried schoolmistress, aged 24, who was admitted on the 29th of December, 1864. She was feeble, and had a strumous appearance, with a hectic flush on each cheek. Her extremities were habitually cold, but there was neither oedema nor varicose veins of the lower extremities. Occasionally she had a troublesome cough, and expectoration was free, especially at night; but there were no night sweats. On percussion, the left side of the thorax was duller than the right, and expiration was a little prolonged in the left lung. Dr. Parson, who examined the chest carefully, thought that there was "no tubercle, unless in small quantities and scattered." The heart's sounds were normal, but the heart was displaced upwards, the apex being felt between the third and fourth ribs. The liver, stomach, and transverse colon were also displaced upwards. The urine was of low specific gravity, about 1015, but contained no albumen. The whole abdomen was occupied by an irregular tumour, in some parts of which fluctuation was perceptible.

The patient's parents were healthy; but three of her sisters had died of phthisis. She herself had always enjoyed good health, and had menstruated regularly up to Christmas, 1863. About that time her body began to enlarge without any known cause; pain in the *left* side became tolerably constant, and occasionally acute. By March, 1864, the swelling was chiefly felt on the *right* side of the abdomen; it steadily increased in size and became fluctuant. In October, 1864, and again in November of the same year, Dr. Robbs, of Grantham, tapped, and on each occasion drew off about twelve pints of clear viscid fluid. After her admission to the hospital in December, a little swelling of the left leg was observed. On the 4th of January, 1865, I tapped and removed seventeen pints of fluid. After the tapping, crural phlebitis in the left side increased, and the leg and thigh were much swollen and very painful. The heart and liver descended a little, and the general health improved; but the cyst refilled rapidly, and on the 30th of January I tapped

again and removed eighteen pints of whitish glutinous fluid, similar to that before evacuated. After this tapping, groups of cysts, irregularly disposed, and evidently adhering in some places to the abdominal wall, were felt filling the whole of the hypogastric region, and on the right of the median line, above the umbilicus, extending nearly up to the sternum.

The uterus was high and to the left side; its mobility was restricted. The os was small and virginal; the tumour was felt to the right side of the uterus, pushing that organ to the left, but the tumour was scarcely below the brim of the pelvis. After the last tapping the heart beat a full inch lower than it had done before; but the apex of the left lung was still duller than the right.

Although the feeble state of the general health, the displacement of the thoracic viscera, and the family history, did not augur favorably for ovariectomy, it was so clearly the only resource that it was performed on the 15th of February, after consultation with Dr. Routh. An incision was commenced one inch below the umbilicus, and carried downwards for five inches: there were extensive adhesions between the cyst and abdominal wall, above and to the right of the incision, extending to the brim of the pelvis, but they gave way to the hand. Having tapped and emptied a large cyst, and broken down a second within the first, the tumour was drawn out, and a piece of adhering omentum was separated. The pedicle was three to four inches in length, extending from the left side of a long thin uterus; it was secured in a small clamp, and left outside without traction. There was a little oozing from the separated adhesions. The blood was carefully sponged away, but no vessel required ligature. The right ovary was felt to be healthy. The wound was closed with five deep and three superficial sutures.

The patient rallied well, complained of but little pain, and only required one opiate. The stitches were all removed on the third day,—the clamp on the eighth day. The bowels acted for the first time on the thirteenth day, but there had been no uneasiness from the prolonged constipation. She

left the hospital four weeks after the operation, and returned to the country in good health.

About twenty-two pints of fluid were evacuated at the operation, and the more solid remainder of the tumour weighed about seven pounds. The following description of this part of the tumour is by the late Dr. C. G. Ritchie.

"The great bulk of the tumour is made up of five or six large cavities, whose dissepiments have been cut through or torn through during the operation. The walls of these cysts are from half an inch to two inches thick, but they owe their thickness entirely to the presence of innumerable vesicles, some of which are of the size of a pea, others that of a pippin. The vesicles are for the most part diaphanous, but in almost every one of them is to be observed a white streak, which examination shows to be contained in the jelly-like contents and not in the translucent wall. Some of the cysts, instead of being diaphanous, are quite white; the contained fluid in these has much the appearance of milk. Some of the cysts are set so closely together that they considerably modify the shape the one of the other; others, again, are solitary and spherical. The outer tunic of the tumour is of course peritoneal; it is marked with traces of inflammatory adhesions. The remains of the pedicle consist of a double layer of broad ligament, of the ovarian vessels, and the Fallopian tube."

The patient remained extremely well for more than a year after the first operation. On the 14th of February last she wrote to me as follows: "A year having elapsed since my operation, I am thankful to tell you that I am quite strong again, and have never taken any medicine since I left the hospital. I am a wonder to myself when I consider how dangerously ill I was." I did not hear of her after this until she came to town and called on me, on the 6th of August, when I found a semi-solid tumour of the right ovary, reaching up to the false ribs on the right side, in the centre to two inches above the umbilicus, and extending towards the left side half way between the umbilicus and anterior superior spine of the ilium. The uterus was freely moveable.

She said she had not noticed any increase in size for more than a month, but had felt pain in the right side in the spring. The catamenia had been regular till a month ago, but latterly had become scanty. At the periods in April and May dysmenorrhoeal pain was excessive. There was some cough, but no very urgent symptom, and she returned to the country to consider my advice to submit again to ovariectomy before her general health became seriously impaired. About a fortnight later, on the 24th of August, her sister wrote to tell me that the patient's cough had become very troublesome, and she was so much weaker, and generally so much worse, that if she continued to lose her strength she would not be able to go through the operation. As the Samaritan Hospital was closed for repairs, a room in the neighbourhood was procured, and the patient came to town on the 29th of August. The tumour had grown very rapidly, dyspnoea and cough were very troublesome, temperature in axillæ 101°, Fahr., and urine scanty. She had begun to perspire a great deal at night. The catamenia were expected in ten days. Careful examination of the chest failed to detect anything not explicable by the displacement upwards of the diaphragm by the ovarian tumour, which just reached the ensiform cartilage. As there was no cyst large enough to tap with any hope of affording even temporary relief, I performed ovariectomy the day after she arrived in town, the 30th of August, 1866, just eighteen months and a half after the first operation. Professor White, of Buffalo, United States, and Dr. Hjort, of Christiania, were present. I was assisted by Dr. Bowen and Dr. Wright, and Dr. Junker administered chloroform. Bearing in mind the slow and imperfect union in my former second operation, when I made the incision very near the cicatrix of the first operation, I made it in this case an inch and a half to the right of the cicatrix (which was exactly in the middle line), and carried it from one inch above the umbilical level downwards for five inches. Its lowest point was about half an inch higher than the level of the lowest point of the cicatrix. Three arteries, one of considerable size, were divided near the lower end of the incision, beneath the

divided muscle, and were tied before the peritoneum was opened. A thin-walled compound cyst was closely adherent all over its anterior surface, but the adhesions yielded easily to my hand. I introduced a large trochar, but the cysts were too small and the contents too viscid for any fluid to escape. I accordingly opened the tumour, broke it up inside, pressed out a great deal of its viscid contents, and then withdrew the remainder, after separating a piece of adhering omentum. A broad thin pedicle extended about two inches from the right side of the uterus. The uterus was in its normal position; but the pedicle of the tumour removed at the first operation passed from the left side of the uterus and adhered firmly to the lower angle of the cicatrix in the middle line of the abdominal wall. The pedicle of the tumour about to be removed was enclosed in a broad clamp, and the tumour was cut away; three omental vessels were tied, and the ligatures cut off short. There was very little bleeding, but as some ovarian fluid had escaped, the peritoneal cavity was carefully sponged out. The pedicle on the left side interfered a little with this process, but it was continued until the sponges came quite clean from the lowest part of the space between the uterus and rectum. Finding that there would be considerable traction on the uterus and broad ligament if the clamp were kept outside, I determined to apply the actual cautery and burn off the portion of cyst left above the clamp, and be prepared to tie any vessel which might bleed on removing the clamp. Protecting the abdominal wall by two shields of talc—a most perfect non-conductor of heat—I used three or four hot irons, and as on separating the blades of the clamp there was no bleeding, the compressed and seared pedicle was allowed to sink into the pelvis. The wound was closed by silk sutures. The fluid or jelly-like substance removed with the fragments of the broken-up tumour, together measured eighteen pints. The following description of the tumour is by Dr. Junker.

“The tumour consisted of an oblong mass, divided by delicate fibro-membranous septa into numerous chambers or loculi of various size. These septa, as well as the main wall,

were exceedingly thin and friable; so much so that the tumour broke up into fragments on very slight pressure. Some portions of the main wall and of the septa were very vascular, and covered with what appeared to the naked eye circumscribed round or oval red spots, having diameters varying from one to three lines. Under the microscope, however, these spots proved to be a dense capillary network, with well-defined abruptly terminating outlines. The interior of the loculi was in many places coated by a true tubercular deposit, often corresponding in size and situation to the red spots just described. In other places the tubercular exudation was more profuse, and some of the lesser loculi were entirely filled by yellow tubercular masses. Genuine tubercles, softening, or in a state of cretification (*Verkreidung*, of Rokitansky), were also found imbedded in the stroma. In some places the septa were softened or destroyed by the tubercles. The loculi were filled with a thin reddish or yellow, slightly ropy fluid, which in some of the chambers appeared more turbid from the presence of minute tubercles suspended in the fluid."

The progress of the patient after the second operation was quite as satisfactory as after the first. There was rather more pain and sickness during the first thirty-six hours after operation, and three opiates were required during the first twelve hours. After the second day all unfavorable symptoms ceased, and she made a most satisfactory recovery. On the second day I left town, and Dr. Junker carried on the after treatment; he removed the stitches on the third day. The wound was well united, but there was an appearance of commencing suppuration around the ligatures left on the superficial vessels. There was no more sickness, and the cough had entirely disappeared. On the sixth day there was some purulent discharge beside the ligatures, but it ceased on the ninth day, when the ligatures came away. The bowels acted on the twelfth day—the pulse, respiration, and temperature by that time having reached the normal standard. Before the operation the pulse was 108, respirations 20, temperature 101·4. The following table from



Dr. Junker's notes gives the range on each day after operation until the bowels acted and the normal standard was maintained.

	Temperature.	Pulse.	Respiration.
Day of operation .	101·4	112	32
First day after .	100·4	98	22
Second „ .	99·5	86	20
Third „ .	100·4	92	24
Fourth „ .	99·0	90	26
Fifth „ .	99·4	88	20
Sixth „ .	100·2	100	24
Seventh „ .	99·2	84	20
Eighth „ .	99·4	78	22
Ninth „ .	98·8	84	20
Tenth „ .	98·2	84	22
Eleventh „ .	99·4	94	24
Twelfth „ .	98·4	84	22

Morning and evening observations were taken, but only the highest range each day has been given in the above table. The fall in temperature, pulse, and respiration after the operation is remarkable and very unusual. The slight elevation on the third day before the removal of the stitches is very common. So is the rise on or about the sixth day when there is any superficial suppuration about the wound, as well as the rise just before the bowels are relieved for the first time. After the bowels acted, strength was rapidly gained, and the patient returned by railway to Lincolnshire twenty-nine days after the operation.

*Note added November 13th, 1866.*—I have heard from her twice since her return home. The last letter is dated November 10th, 1866. She says, "I think upon the whole I feel as well as I did after my first operation. My voice is stronger. I can sing the upper notes with greater facility than formerly. I can sing from A up to C natural." I was curious to have the range and power of the voice observed after the removal of both ovaries, and it could be done with unusual accuracy in this case, as the patient is a teacher of singing.

Many circumstances mentioned in the history of this case

are suggestive of some remarks upon the relation between tuberculosis and ovarian diseases; but this subject is of too much importance to be treated incidentally, and I shall therefore conclude by a few remarks upon the comparative frequency of the disease in one or both ovaries, and on the appearance of disease in one ovary after the other has been removed.

Some writers on ovarian disease have asserted that the right ovary is much more frequently diseased than the left, and that coexisting disease of both ovaries is extremely rare. But, on examining the grounds for these assertions, we find that they are principally based upon examination of patients during life, or patients who have not been submitted to ovariectomy.

When we come to examine the result of post-mortem examinations we find (as a very little reflection would lead one to expect) that, as there is no anatomical or physiological reason why the right ovary should be more frequently affected than the left, so, in fact, one ovary is found to be diseased as often as the other.

Of 80 cases collected by Dr. West from Scanzoni, Lee, and his own notes of post-mortem examinations, in 28 the disease was on the right side, in 26 on the left side, and in 26 both ovaries were diseased—so that in about one third of the cases both ovaries were diseased. In 1865 Scanzoni again drew attention to this subject in the Würzburg '*Medicinische Zeitschrift*.' In a paper '*On the Relation of Disease of both Ovaries to the Ovariectomy Question*,' he gives the result of an examination of the reports of post-mortem examinations for the previous fourteen years by his colleagues Virchow and Förster. These records were examined with the sole object of ascertaining in how many cases one or both ovaries were diseased—and in 99 cases of ovarian disease it was found that in 48 one, and in 51 both ovaries were diseased—so that in more than half the disease was on both sides. The tendency to disease of both ovaries appears to be greater before the age of fifty than in older women. Of 52 women under fifty, both ovaries were diseased in 31 ;

one ovary only in 21 (59 per cent. to 40); of 44 women above fifty, both ovaries were diseased in 17 only, while one ovary was diseased in 27. Thus, under fifty, we had both ovaries diseased in 59 per cent.; above fifty, only in 88 per cent.

But it must be remembered that any conclusion drawn from post-mortem examination would in all probability differ very widely from results observed in ovariectomy. The first series of facts shows what may be expected when ovarian disease has proceeded to its natural termination, or has only been modified by palliative treatment. The other series shows what may be expected when the patient is subjected to radical treatment before the disease has advanced to its latest stages. All observation tends to the conclusion that disease begins in one ovary and advances to a considerable extent in that ovary before the other is affected, and that in about half of the cases it proceeds even to its fatal termination without any disease occurring in the opposite ovary.

If, then, in only about half of the cases where ovarian disease has reached its *latest* stage, disease of both ovaries is found, we might expect that in *earlier* stages of the disease both ovaries would be much less frequently affected; and, so far as my observation has gone, this is the fact. In the first 150 cases in which I performed ovariectomy I only removed both ovaries in seven cases. In three other cases the ovary not removed presented some indications of disease in a very early stage, but not sufficient to warrant its removal.

It is not improbable that in some of the earlier cases slight disease of the opposite ovary may have been overlooked; but, making every reasonable allowance for such error, it is not probable that when ovariectomy is performed both ovaries will be found diseased in more than 8 per cent. of the patients. Scanzoni thinks that as both ovaries have been so seldom removed (he finds only 25 on record), operators must either have overlooked disease of the second ovary or thought it insignificant, or believed that the removal would add too much to the danger. Of 25 recorded cases 11 only recovered, and 14 died, a mortality of 56 per cent.;

whereas, of 468 cases, where only one ovary was removed, the mortality was only 44 per cent.

We require additional facts before we can estimate the increased risk added by the removal of the second ovary. I may just mention that of the 7 cases just mentioned, 4 recovered and 3 died.

As to the frequency with which, after successful ovariectomy, the ovary not removed, but examined and found healthy, becomes diseased, besides the case just related three others have come under my notice.

In my second case, operated on in 1858, the patient remained well for seven years. Then disease of the opposite ovary appeared, so evidently of a malignant character, that no operation was thought of, and soft cancer was found after death.

In the third case, also operated on in 1858, the patient died of peritoneal cancer ten months after operation, and disease had commenced in the remaining ovary, which was enlarged to the size of an apple.

In my 43rd case, operated on in 1862, disease of the opposite ovary came on two years afterwards and was treated successfully by vaginal tapping and drainage. The patient is now well. I have not heard of any other of my patients in whom disease of the second ovary has appeared after successful removal of the first.

Sometimes during an operation, after removal of one ovary, some slight alteration in the other may be observed, and the question of removal of the second ovary may arise. In more than one of my cases this question has arisen. In narrating the 112th case of ovariectomy in the first volume of my work on 'Diseases of the Ovaries,' after recording the removal of the right ovary from a young lady, aged 19, I continue, p. 307.

"The left ovary was enlarged to nearly double the normal size. Two follicles, about the size of cherries, were distended by clot. These I laid open, turning out their contents. . . . The operation was peculiar on account of the doubt as to the treatment of the left ovary. I resolved after con-

sulting with Dr. Greenhalgh (who was assisting me) not to remove it, because—

“*a.* The ligature which would have been necessary would have added seriously to the risk of the operation.

“*b.* It is not certain that *disease* was present in the ovary, or that it would progress, and if it did a second ovariectomy could still be done.

“*c.* It seemed hard to unsex a girl of nineteen. Perhaps the clots might have been left alone, but turning them out could do no harm, and might do good.”

This operation was performed in November, 1864. This patient recovered well, went into the country four weeks after operation, was married in October, 1865, and is now the mother of a child, born in September last, twenty months after operation, and eleven months after marriage.

The age and conjugal condition of the patient, and the amount of disease in the ovary, must of course be the chief guide to the surgeon in inducing him to leave or remove an ovary in any doubtful case.

When a surgeon has removed a large diseased ovary and the woman recovers, he has in very many cases the great satisfaction of feeling that his patient has been restored to perfect health. Experience has proved that the remaining ovary generally carries on its functions, and that the woman may become the mother of healthy children of both sexes. The patient is not mutilated as by the amputation of a limb, nor does the general health suffer as it frequently does after the greater amputations.

There certainly is nothing like the tendency to recurrence which there is after the removal of malignant tumours, probably by no means so frequent occurrence of disease elsewhere as after successful ligature of a diseased artery, or disease of the opposite lens after successful removal of one cataract, or formation of a second calculus after a removal of one by lithotomy or lithotrity; and certainly no such prolonged suffering as the chronic cystitis which not unfrequently follows these operations.

The rule is that by a successful ovariectomy the patient is

restored to a state of health so perfect that she and her friends are as surprised as they are gratified. But there are exceptions to this rule. In some cases a disease believed to be innocent proves to be malignant, soon recurs, and proves fatal within a few months, or even within a few weeks after apparent recovery. In other cases the ovary which is left untouched because it is believed to be healthy, or so slightly diseased that its removal is uncalled for, becomes the seat of disease. In what proportion of cases this occurs we have as yet no means of knowing. It is only within the last ten years that the operation has been performed sufficiently often to furnish data for reliable statistics, and it is difficult to ascertain, even in some of these later cases, what has been the state of the patient's health for some years after operation. But it would be unreasonable to expect that in all cases the ovary left in the body would remain healthy. It is for future observation to decide how often and in what class of cases a recurrence of disease ~~may be feared.~~ It is satisfactory, however, to learn that if the remaining ovary should become diseased, the first operation need not add much to the difficulty of the second, and that of four cases in which a second ovariectomy has been performed, two have proved successful.



SEQUEL TO A CASE  
OF  
COLOTOMY FOR VESICO-INTESTINAL  
FISTULA

RECORDED IN THE LAST VOLUME OF THE 'TRANSACTIONS.'

DEATH HAVING ENSUED FROM AFFECTION OF THE  
BOWEL HIGHER UP.

(ACCOMPANIED BY A PREPARATION AND DRAWING OF THE  
PARTS CONCERNED IN THE DISEASE.)

BY

TIMOTHY HOLMES, F.R.C.S.,

SURGEON TO THE HOSPITAL FOR SICK CHILDREN; ASSISTANT-SURGEON  
TO, AND LECTURER ON SURGERY AT, ST. GEORGE'S HOSPITAL.

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Received Nov. 2nd, 1866.—Read Jan. 5th, 1867.

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THE object of the following paper is to lay before the Society the termination of a very rare and highly interesting affection of the intestinal tube, by which a communication was effected between the sigmoid flexure of the colon and the bladder, leading to great distress from the passage of *faeces per urethram*. On this account colotomy was performed in June, 1865, and with immediate and permanent relief to the symptoms, as related in the last volume of the 'Transactions.' The diagnosis made of the case at the time was that the fistulous communication was formed between



the sigmoid flexure of the colon and the bladder, and that it was the result of simple or non-malignant ulceration. Both conclusions, it will be seen, were justified by the post-mortem examination. But after an interval of about fifteen months the fæces reappeared in the urine, and shortly afterwards they altogether ceased to pass by the artificial anus. It was feared that the ulceration had extended from the sigmoid flexure into some adjacent coil of small intestine accidentally adherent. But on post-mortem examination it was discovered that the cause of death was a similar, though independent, affection of the cæcum.

The following particulars will complete the notes of the case :

*Case and post-mortem examination.*—After the operation the patient recovered completely from the consequences of the intestino-vesical fistula, except that he still occasionally, and at rare intervals, passed some water per anum. This, however, did not amount to any serious inconvenience. The urine was quite clear and free from fæces.<sup>1</sup> The man improved greatly in health and strength, and talked about soon resuming his occupation. He still, however, suffered from occasional attacks of pain—referred to the pelvis. These pains became more severe in the latter part of the summer of 1866, and he began rather to fail in health and to lose flesh; but there did not seem any serious cause of alarm

<sup>1</sup> It was so on all the occasions that I procured any for examination; but it was said that he occasionally passed fæces per urethram. If this were really true, which seemed doubtful, it was at such rare intervals, and in so small quantity, that it might have been merely the result of some small masses accidentally slipping past the lumbar opening. On this subject Mr. Churchill, Mr. Faithorn's assistant, who was in attendance on the case, gives me the following information:—"The urine was perfectly free from fæces till the end of June, 1866, when a little fæcal matter passed; and then it remained free from fæces till a month before his death. At the end of August or beginning of September the water was examined and found highly purulent, but free from fæces. After the pus disappeared from the water, the fæces passed continually per urethram. No fæces were passed by the lumbar opening for the last five days of life."

until the month of September, when fæces again made their appearance in the urine, and this now became permanent. After about three or four weeks it was noticed that the artificial anus no longer transmitted the fæces, which passed entirely by the bladder. The urine also began to pass in much larger quantity through the anus, and his sufferings became very great indeed, more so than before the operation. He lost flesh rapidly, could hardly take any food, and after lingering in much distress for some time he died on October 26th.

After death Mr. Faithorn was so kind as to remove the left kidney, bladder, ileo-cæcal portion of the intestine and the whole of the large intestine down to the anus, in their natural connections, as they are now exhibited to the Society. The artificial anus is seen to be just below the lower end of the kidney, at the junction of the transverse and descending colon, and its relation to the peritoneum is well shown.

On laying open the bladder, a large irregular opening is seen by which it communicates with the large intestine. There is, in the first place, a free communication with the sigmoid flexure of the colon, which portion of the gut is drawn down to the fundus of the bladder and opens directly into it. The gut is somewhat bent at an angle in this situation, and thus considerable constriction or obstruction may at one time have been produced. But there is no appearance of malignant or other deposit in the tissue of the gut. External to the opening between the colon and bladder, and lying in the cellular tissue of the pelvis surrounding the gut, is a large lump or tumour. This has been laid open from behind in dividing the coats of the gut. It displays, on naked-eye and on microscopical inspection, no trace of malignant deposit; but, on the contrary, the thickened fibrous tissue and fat looked perfectly healthy both under the microscope and to the eye. This lump was obviously the result merely of healthy or ordinary inflammation, caused by the irritation of the disease in the gut and the passage of fæces into the cellular tissue. The cæcum was also closely adherent to the fundus of the bladder, and a similar perfo-



at the time of my receiving the parts the serous coat given way; but it appeared to me as if this had taken place in handling the parts, and, on reference to Mr. Faithorn, I found that this was the case. Around this ulcer the coats of the gut were thickened, and its calibre contracted; but, on microscopic examination, no cancerous elements could be detected in the thickened portion, nor was there any appearance of cancer to the eye. That there had been no real cure of the gut here was proved by the fact that below the ulcer it was full of feces. The bladder also was completely full of feces, which had passed into it from the colon. The only enlarged gland that could be discovered in the omentum. It was about the size of a hazel-nut, and contained here and there a drop of pus, but no solid matter.

#### EXPLANATION OF WOUNDS

- A rod passed from the original vesical opening into the upper part of the colon.
- A second rod passing from the subsequent vesical opening into the colon near the ileocolic valve.
- A rod passed from the artificial anus into the colon just below the ileocolic valve.

**Remarks.**—Although this case terminated in disappointment as concerns the restoration of the patient to enjoyment of life, yet I think that it only the more fully confirms the views which I advocated in the case published in the last volume of the

existed before the operation, and that the opening in the cæcum was formed afterwards, shortly before death, and was the cause of the failure of the artificial anus, and the miserable death which followed. It seems almost superfluous to point out, that if the perforation in the cæcum had existed before the operation, the latter would never have relieved (as it did for more than a year) the passage of fæces into the bladder. But this is perhaps too clear to need discussion. A more interesting problem is the nature of the ulceration. I am myself strongly of opinion that there is nothing specific about it; that it is neither cancerous nor tubercular, but the effect of simple inflammation, just as an ulcer on the leg might be. We see such ulcers often in the stomach, where they frequently cause sudden death by perforation. Why not in the intestine? In the œsophagus non-malignant ulceration is also common, as the 'Transactions' of the Pathological Society will show, where five cases are recorded, viz. vol. ii, p. 208; iii, p. 316; vi, p. 179; viii, pp. 175, 191. In the second of these cases the ulceration had perforated the tube and formed a communication with the pericardium; and it may be a matter of interest to notice that in the last or fifth case the patient had repeatedly suffered from abscess between the bladder and rectum. Again, we often see fæcal abscess in various parts of the intestinal tube, which is only remotely and dubiously connected with any constitutional condition, and which leaves the patient in sound health after its healing, if he can bear the irritation of the disease. My case appears to have been of this kind. Its interest pathologically lies in the occurrence of multiple ulcers in the gut, and in the singular fact that two of them should have reached the bladder, and its surgical interest in the complete success of the operation in relieving the symptoms connected with the affection as it at first showed itself in the sigmoid flexure of the colon.

**TWO CASES**  
**OF**  
**PERIODICAL INFLAMMATION OF THE**  
**RIGHT KNEE-JOINT ;**  
**WITH REMARKS.**

**BY**  
**CHARLES H. MOORE, F.R.C.S.,**  
**VICE-PRESIDENT OF THE SOCIETY ; SURGEON TO THE MIDDLESEX AND**  
**TO ST. LUKE'S HOSPITALS.**

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FOURTEEN years ago I had under my care a female patient, who was attacked with inflammation of the right knee-joint at almost regular intervals of time. After continuing, on each occasion, for about the same number of days, the attacks were followed by a state of health, the successive periods of which were also of nearly the same duration. In the course of many years the rate at which the disease was going, so to speak, became accelerated, but, as the recurrence of many successive attacks was nearly punctual, and could be predicted almost to an hour, and as, moreover, they arose without apparent external cause, it seemed clear that the whole disease was periodical, and I published the case under that designation.

It was not without hesitation that I ventured to put it forth as a fact. For the records of medicine contain few examples of a periodical repetition of local diseases, and none resembling that which I had observed. Three cases of regular attacks of vomiting, having a definite duration, and separated by equal intervals of health, are to be found in the 'Transactions of the Medical Society of London,' 1817, vol. i, p. 89. And the 'Medical Notes and Reflections' of Sir Henry Holland contain several examples of diseases presenting a periodical character. Some of these I may briefly refer to in confirmation of the remarkable cases I wish to record.

Dr. Joseph Adams's first case was that of a young gentleman, who was troubled for several years with a periodical sickness, which occurred every tenth day, and after lasting for several hours subsided. During the succeeding nine days he always remained tolerably well, and on every tenth day the attack returned. He was cured by arsenic.

The second case occurred in a lady, who had very painful nausea and sickness, lasting throughout the whole of every seventh day. With scarcely any variation, this disease recurred regularly on the seventh day for fourteen years, when it was cured by arsenic. During these fourteen years she produced a child almost yearly, and neither gestation, parturition, nor attempts at suckling, made any difference in her complaint.

The third was a woman who for eighteen years had a weekly periodical sickness and headache at uncertain periods. The former was temporarily relieved by arsenic, which, however, did not stop the headache. Being a dispensary patient, she did not continue under observation to the end of her ailment.

"Dr. Leonhard, of Muhlheim, relates a singular case of a woman, in whom an attack of influenza (the severe epidemic of February and March, 1837) passed into a local quotidian intermittent, affecting first the left, afterwards the right arm, with every successive symptom of regular ague; the rigor, heat, and stage of perspiration all distinctly marked in each fit—other parts of the body wholly unaffected—the disorder

cured by quinine.”<sup>1</sup> Sir Henry Holland, moreover, relates instances of severe pain in the upper jaw, coming on each day precisely at the same hour, of daily painful enlargements of the tonsils; of daily punctual attacks of ophthalmia; of cough recurring at one hour only in every twenty-four; and of urgent thirst affecting a patient at a regular hour on alternate days. The character of the disease in most of these cases was further attested by their being benefited by quinine.

I am quite aware that the return of an ailment, at a given hour or day, does not determine the character of periodicity to be an essential part of its nature; and the semblance of such regularity which some cases present, may be explained by an accurate observance of their circumstances. Thus, in February last, I removed the tongue at the level of the anterior pillar of the fauces, together with the floor of the mouth, on account of an extensive ulcerated cancer. After the healing of the wound, I was disappointed to find, at each afternoon visit, symmetrical, nearly globular, firm, and painful swellings of considerable size in the digastric triangles, and I feared that a rapid growth of glandular cancer was proving the Surgeon to have done rightly, who had allowed the disease to go on to so great an extent unchecked by an operation. After some time, however, it came out that the swellings were not always present; then, that they arose while the patient was taking his midday meal, and subsided in the course of the afternoon. And it was soon clear that they were of the nature of mumps, and were due to contraction of the scar at the divided orifices of the Whartonian ducts, whereby the out-flow of the saliva, secreted during dinner, was obstructed. This explanation of the occurrence is borne out by the continued health of the patient to the present time.

My conviction of the reality of the disease in my first case was confirmed last year by my meeting with a second example of inflammation of the right knee-joint, presenting the same peculiar character of periodicity. Having thus had the opportunity of twice recognising this unusual occurrence, and having taken such precautions as convinced me that the

<sup>1</sup> ‘Medical Notes and Reflections,’ 3rd edition, p. 293.



symptoms were not feigned or artificially produced, I venture to introduce both cases to the notice of the Society.

**CASE 1.**—A slender and delicate woman, æt. 43, came under my care in the Middlesex Hospital, December 30th, 1852, with her right knee-joint loosely swollen with fluid. The next day the fluid was almost gone, and by January 1st, 1853, it had quite disappeared. The joint had begun to swell on December 27th.

Until January 5th, the joint was quite well. On the 6th, she felt her right foot cold two or three times, and in the course of the day a little swelling appeared in the right knee. On the 7th there was a considerable but loose swelling and some increase of heat about the knee. She felt otherwise quite well, and her tongue, pulse, appetite, and evacuations were natural. On the 8th the synovial membrane was quite full and tense, bulging a good deal on each side of the rectus tendon, and reaching three inches above the patella. On the 9th the joint was loosely swollen again, and on the 10th was well. There was a movable body in the outer part of the joint, but it gave her no inconvenience. She appeared and felt well, but she had a little enlargement of the spleen.

Her account was that at the age of 18 she had ague for six months, which was at first quotidian, then tertian, and afterwards quartan; and that it ceased on her taking a powder which caused copious perspiration. All her family, with whom she was then living at Dover, suffered from ague.

She married and had some children, and her health was good for eight years after the cessation of the ague. She then, eighteen years ago, found her knee become painful at the close of a busy day at household work. The joint swelled, and the swelling increased until the third day, when it subsided. She remained well until thirty days after this attack, when it was repeated, lasted precisely the same time, and then again left her perfectly well. The inflammatory swelling recurred for some considerable period at regular intervals of a month, but afterwards it was repeated more frequently, returning every three weeks. About seven years

ago, it began to reappear every ninth day, varying only in the hour of its onset, but not in the day. The attack was always preceded by coldness of the foot, and there was always a moderate degree of pain on the third day, which was increased by previous use of the knee, but lessened by repose.

She said that she had had several children since, as well as before she became subject to these recurring inflammations, and that her youngest child was about six years old. The attacks had ceased in every pregnancy at the third month, and had not returned till the third month of lactation, when they recurred regularly as before. In point of time the menstruation was independent of the attacks; it had always been healthy, and had always occurred during lactation, equally during the three months in which the attacks were interrupted as in the later period. No other circumstance but pregnancy and lactation had exerted any influence upon the ailment.

In expectation of the attack on the evening of the 14th, I ordered a calomel and colocynth pill for the 13th, and a senna draught on the following morning. Three grains of quinine were also taken two or three times during the 14th, and some ice was applied to the knee. The attack failed to make its appearance at the expected time after the adoption of these measures, and there seemed to be some hope that it had been averted. After a few hours' delay, however, it returned, and it proved to be so violent that she said she had never had so much pain on any previous occasion. The joint became more than usually swollen, but it entirely subsided again.

After this I prescribed arsenic to be taken internally, but it failed to check or to alter the course of this singular affection.<sup>1</sup>

CASE 2.—Susannah R—, æt. 21, a housemaid, well nourished, florid and healthy in appearance, came into the Middlesex Hospital under my care January 3rd, 1865. Her right knee-joint was loosely swollen with fluid, which occupied

<sup>1</sup> From the report of one of my Clinical Lectures in Surgery. The 'Lancet,' 1864.

the synovial cavity above and on either side of the patella. The joint was not tender or painful, and it was reported to be recovering from an attack of swelling and pain, which had commenced spontaneously December 30th.

On the following day the right knee was no longer swollen, and both it and the left measured  $13\frac{1}{4}$  inches in girth at the top of the patella. On the 5th the right knee was a quarter of an inch less than the healthy one.

The patient stated that this inflammatory attack was one of a series which recurred regularly every twelfth day. Each attack lasted five days, and laid her up, but in the intervals she was quite well and able to do her work.

On more precise inquiry it was ascertained that she first felt pain and stiffness, with swelling, of the right knee three months ago. She applied wet cloths, and the next morning felt nothing more of it. A similar attack came on about twelve days afterwards; but this was more severe, the pain and swelling being greater. After twelve days more the knee inflamed again. She did not remember the dates of these three attacks.

A fourth attack occurred November 23rd, and a fifth on the 5th of December. This attack lasted five days, and she left her place and became an out-patient under the care of Mr. Lawson, at the Middlesex Hospital. From this time she rested her knee.

The sixth attack was delayed to the 13th day, not beginning until December 18th. It was less severe than most of the previous ones, continuing only three or four days. On the 29th the catamenia appeared, and they were perfectly natural.

The seventh attack began on the 12th day, December 30th. It was subsiding at the time of her admission into the Hospital, and it lasted five days.

With the exception of the sixth, which was unpunctual and of short duration, each of these attacks was more severe than the previous one. They usually began during the night or early in the morning with shooting pain in the leg and thigh; this was followed by a dull aching pain in the knee,

and at the same time by swelling of the joint, which progressively increased for two days and a half. The swelling was then at its height, and the pain so irksome that she could not keep the limb in any posture, and was restless the whole night. The pain after this moderated and, with the swelling, gradually subsided, and was quite gone on the fifth day. She noticed redness of the knee, and thought it brightest during the decline of the disease. She had not felt coldness of the limb or general shivering at the beginning of the attacks, nor local perspiration during their course. And she had not been conscious of any feverishness before or during the inflammations. She sometimes had pains in the right arm while the right knee was painful, and in the left knee during the intervals of the periodical attacks, but no swelling or stiffness or other pains of any joint except the right knee.

*Family history.*—Her father is a tailor, alive and healthy; her mother died four years ago of phthisis. Three of their eight children died in infancy, and a fourth died in the hospital twelve years ago of cancer of the left knee and the brain. One of her sisters had swelling of one knee, which was cured by a mustard poultice and did not return. Another sister is now consumptive.

*Previous history.*—She was born in Somers Town, and always lived in London. Her health was good until two years and a half ago, when she resided in the Regent Circus, and had debility, nervousness, bad appetite, dyspnœa, and pain in her right side. In February last she went to live in Kensington, and her health much improved. She never had ague, nor had any one living in the same house with her, although in the neighbourhood of her residence at Kensington many new houses were being built, and the earth was a good deal turned up. She herself attributes her illness to kneeling on stone steps, the washing of which was a new duty undertaken by her not long before the first attack of inflammation. Her catamenia are said to have been always natural and regular, at periods independent of the times of the attacks; they last occurred on the 29th of December, the day preceding the present attack. Her

resting during the last five weeks, and her removal from Kensington, have not put a stop to the attacks.

From the 5th to the 10th of January the knee was observed daily, but nothing unnatural was discovered in it either as to temperature or measurement. It was in both respects about equal to the left knee. A loosely attached body, or thickened portion of the synovial membrane, could be felt to move under the fingers in the space between the vastus externus and the anterior edge of the fascia connected with the tensor fasciæ femoris, where the joint is most superficial.

*Eighth attack.* Jan. 10th.—Some aching was felt towards evening in the right knee, and about midnight severe shooting pain. In the latter part of the night she slept well; but, on awaking in the morning of the 11th, she found herself unable to move that knee without pain. In the forenoon the knee was found three quarters of an inch larger than the left. The swelling was soft and fluctuating, and the skin about the knee was reddened. The pain varied in severity, and was less than it had been in the night. She did not observe any chilliness to precede the attack, but its anticipation by a day caused this symptom not to be looked for. After her dinner of meat and porter she was flushed, and her pulse rose to 100, and was full and bounding; her skin was hot, and the palms of her hands moist. The temperature of the right knee was  $95\cdot25^{\circ}$ , that of the left  $89^{\circ}$ ; both knees were rolled with a flannel bandage. At midnight the heat of the right knee was at  $94\cdot5^{\circ}$ , whilst that of the left was  $93^{\circ}$ .

12th.—Except when disturbed at midnight she slept from 10 till 5. To-day the right knee is less painful and less tense; its temperature is  $93\cdot3^{\circ}$ , that of the left being  $90^{\circ}$ , and its circumference is  $13\frac{7}{8}$  inches, which is half an inch larger than the girth of the left. After dinner her pulse was 86, not full or bounding; her face was still flushed, and her hands moist, but less so than yesterday. The bowels have been open daily; the tongue is clean and the appetite good; the urine appears natural.

13th.—She slept well last night, and she is less flushed to-day; her hands are cool. Pulse 84. Some dull aching pain

continues in the right knee, but it has much moderated, and the swelling appears to be gone. The right knee is  $13\frac{1}{2}$  inches in girth, being not one quarter of an inch greater than the left. In temperature the right knee stands at  $94.1^{\circ}$ , the left at  $93.1^{\circ}$ . By 8 o'clock in the evening all pain in the joint had ceased.

14th.—She slept well and is entirely without pain to-day. The girth of the right knee is  $13\frac{1}{2}$  inches, being somewhat less than that of the left, and its temperature,  $92^{\circ}$ , is also below that of its fellow, which is  $92.3^{\circ}$ .

Her knee now felt well, and she was up and about; flannel rollers were kept constantly on both knees, and their temperature was examined daily until the 19th, and found to be equal, and varying from  $92^{\circ}$  to  $90^{\circ}$ .

On the 20th, the temperature of both knees was depressed, that of the left much more so than that of the right. The right knee was at  $89^{\circ}$ , the left at  $86^{\circ}$ ; there was, however, neither pain, redness, nor swelling of the right knee; she said merely that the right knee felt hot. On the 21st the temperature of the right knee was  $93^{\circ}$ , and was a degree and a half above that of the left. There was, however, no difference between them in her sensations.

*Ninth attack.* 22nd.—The attack returned this evening about 6 o'clock. She did not feel coldness of the knee, and did not shiver, but first noticed that the bandage became tight. In half an hour pain came in the right knee, and gradually increased. It awoke her occasionally in the night, but did not keep her awake.

23rd.—The joint is swollen, and about a quarter of an inch larger than the left; the synovial membrane bulging especially on the outer side of the patella. There are marks on it resembling scratches. The pain is less severe than in the last attack. She had pain during the night in the right side of the chest, and it continues to-day. Pulse, after dinner, 96. The hands are not moist.

24th.—She was awake last night and kept awake for an hour and a half by severe shooting pain in the right knee, after which she slept again for four hours. To-day the pain

has moderated, and is as slight as it was yesterday. The swelling has not increased, and there is no tenderness. Pulse 84. She is not hot or feverish. Bowels are open.

25th.—She slept well. To-day she has still less pain in the knee; the temperature of it remains high, though the swelling is moderate. She has a cold, with inflamed left tonsil, and her pulse is 100.

26th.—The pain and swelling of the knee are gone, but still the temperature of it remains high, and her pulse 96. The throat is less inflamed.

27th.—Although there is no swelling of the knee there is a slight shooting pain in it. Its temperature is  $95.3^{\circ}$ , a degree above that of the left knee. The throat is well, and she feels well in herself, though she has dull pain in the right side.

28th.—The temperature of the two knees are equal,  $94.2^{\circ}$ .

29th.—The catamenia appeared.

30th.—Right knee,  $93.2^{\circ}$ ; left knee,  $92.4^{\circ}$ . No pain.

31st.—Right knee,  $90.3^{\circ}$ ; left knee,  $89.2^{\circ}$ . She had some aching in the right thigh, from the ilium to the knee, last night, but there is neither swelling nor pain in the knee to-day. The catamenia continue free and natural.

February 1st.—The aching of the thigh continues, but the knees are equal in measurement. The temperature of the right is at  $95.2^{\circ}$ , that of the left at  $94.1^{\circ}$ . The catamenia were ceasing. In the night some aching came on in the left knee, but it did not enlarge. Pulse 68.

2nd.—Right knee,  $97^{\circ}$ ; left,  $97.1^{\circ}$ . Only the aching of the right thigh continues. In anticipation of the expected attack to-morrow night, and to remove the doubt as to the nature of the case, which was suggested by the appearance of scratches on the last occasion, I directed the limb to be encased in a stiff starched bandage, from the middle of the thigh to the middle of the leg.

3rd.—The aching of the right thigh continues, but the knee, though feeling hot, is easy. There is occasional shooting pain to-day in the right arm. Pulse 72.

*Tenth attack.*—About 6 p.m., on the 3rd of February, she

felt the bandage become very tight, and the right knee ached severely. The splint was removed, and the knee was found swollen; the removal of the splint relieved the severity of the pain in the knee. At 7 p.m. one fifth of a grain (by error for a grain and a half) of quinine was injected under the skin of the leg. In the night the knee was hot but not tender, and half an inch larger than the left. There was pain in her right side both in the body and face. Pulse 84. A grain and a half of quinine in solution were injected.

4th.—From the time of the second injection all her pain was much relieved, and she slept a couple of hours in the night. There being smarting at the seat of puncture the injection was diluted to the strength of a grain of quinine in twelve minims of water, and employed three times a day.

5th.—Slept well. Some moderate pain continues in the swollen right knee, but it is less than is usual at this period of the attack, and her chief complaint is of the tenderness of the parts which were injected with the stronger solution of quinine; the other parts are not tender.

6th.—The pain in the knee and the swelling are subsiding. She had a violent frontal headache for three hours last night. She still complains of the tenderness of the first injected parts.

7th.—The swelling nearly gone. The pain is also less, though it has been sharp at times this morning; less tenderness of the injected parts. The last injection was given at midnight of 7th and 8th. On the 8th all pain and swelling were gone from the knee, and she had headache which seemed attributable to the quinine.

From the 9th to the 15th three minims of Fowler's arsenical solution were administered thrice a day, and on the 15th, as the eleventh attack was approaching, the dose was increased to five minims. It was ordered also that two grains of the solution of quinine in twenty-four minims of water should be injected into the limb before the next expected attack should supervene.

*Eleventh attack.*—This took place on the 15th of February, and it was moderate both in pain and swelling. Two grains



of quinine were injected at 4 p.m., and two again at 9 p.m.; she slept well. On the 16th she had aching down the leg, but little pain in the knee. Two grains of quinine were injected about 11 a.m., and at 3 p.m. she was flushed, and had headache, with pain in the right arm and side of the face, as well as in the right thigh and leg. The knee-joint, however, though swollen, was not tense or tender. Her pulse was 104. The headache was gone on the 17th, and through that day and the 18th the pain and swelling of the joint diminished; on the 19th they had entirely disappeared. Two grains of quinine were injected on each of the latter days. On the 11th the dose of the arsenic was reduced to two minims. With the exception of the 20th, on which day she was feverish and had a metallic taste in her mouth, she continued to take the medicine in the reduced dose until the twelfth attack. Some pain and doubtful swelling in the knee came on on the 21st and 22nd, but they soon passed off again. The catamenia recurred on the 25th and were perfectly natural.

*Twelfth attack.*—The joint swelled a little on the morning of the 27th, and was less again in the middle of the day. Some further swelling came on towards evening, and it was attended with slight pain. The temperature of the knee, however, was not unusually high, and but little above that of the opposite knee. Two grains of quinine were injected at midday, and in the evening. About this time the catamenia ceased. On the second day, the 28th of February, there was but little pain or swelling, and she felt well. Quinine was injected but once. Both pain and swelling were gone on the 1st of March, and on that day no injection was made.

*The thirteenth attack* anticipated by three days.—On the evening of the 8th the right knee again swelled, but it decreased on the morning of the 9th, when she was found to be a little feverish, and to be suffering from a cold and cough. Some fluid was distinctly felt in the knee in the afternoon, and the joint was uneasy when moved. On the 10th the right knee had become fuller, and was raised and soft on the inner side. Some moderate pain began in the morning. The joint remained equally swollen on the 11th.

On the 13th the swelling was gone, and there was no pain. The temperature on the 9th and 10th scarcely exceeded that of the left knee; on the 11th the right rose to  $97^{\circ}0$ , while the left, which was uneasy though not swollen, was at  $91^{\circ}5$ . On the 18th the right was  $97^{\circ}5$ , the left,  $95^{\circ}$ . The treatment during this attack was limited to an injection of two grains of quinine on the 12th, and one grain on the 13th.

As it appeared that the quinine injections to some extent controlled the severity of the attacks, I gave up the use of the arsenic, and directed that the injections should be made daily until the time of the next attack. Each injection made her very uncomfortable, producing headache, and sometimes vomiting. The last injection was made on the evening of the 18th.

*Fourteenth attack.*—This began with slight pain in the evening of the 19th. On the 20th there was very slight effusion into the joint, but no pain. An injection of quinine was given. The attack subsided through the 21st.

During this interval injections of half a grain of quinine were given until the 30th. On that day she vomited a greenish bitter matter, and complained of headache, but the knees were natural. The effervescing mixture of Citrate of potash was given every four hours.

*Fifteenth attack.*—On the 31st she had some uneasiness in her knee, but no swelling, and she was flushed and feverish. A little swelling and some slight pain were said to have occurred on the morning of April 1, but neither continued in the afternoon. On the 2nd she was quite well and was up again. The temperature was not taken during this attack.

To avoid the annoying sickness and headache produced by the injections of quinine, I ordered her a mixture, containing one grain of it, to be taken twice daily through the next intermission. She began the medicine on the 3rd of April, and continued it without discomfort until the 7th, when it was interrupted on account of catarrh. She took it again from the 10th to the 13th.

13th.—The *sixteenth attack* being expected, I ordered three grains of quinine to be taken twice during the day.

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No swelling or uneasiness of any kind took place, and she remained until the 17th perfectly well. On that day she was discharged, with the direction to return to the hospital in case of a recurrence of the ailment. She left in good health, florid and stout. The movable body on the outside of the synovial cavity could still be felt, and it seemed a little larger than when she was admitted to the hospital. It gave her, however, no uneasiness whatever, and she did not walk lame.

25th.—On this day, the day for a renewal of the attack, she said she felt a slight weakness in the knee, and that it was a little swollen; but the discomfort had not prevented her walking to the hospital, and no swelling was apparent on examination. She was otherwise very well, and she had not yet returned to her house-work.

During the following four months she paid numerous visits to the hospital, and she reported a slight attack on May 7th, which lasted to the morning of the 9th; an interval in which two attacks missed; a recurrence on June 12th, which was punctual on the forty-first day; and later attacks thirteen days, seventeen days, fourteen days, and fifteen days in succession. These attacks were all slight, except the last but one, which was severe. Since that of August 6th, which was the last, I have not seen her, but have occasionally heard from her that she has had no further attacks.

*Table of attacks, and of temperature and measurement of knee-joints.*

Date.	Right knee.	Left knee.	Measurement of right knee.	Number of attack.
January 4 .....	...	...	13 $\frac{3}{8}$ inches.	VII.
" 5 .....	...	...	13 $\frac{3}{8}$ "	
" 7-9 ...	...	...	13 $\frac{1}{4}$ "	
" 10 ...	...	...	.....	VIII.
" 11 ...	95°-25	89°.	14 $\frac{1}{8}$ inches.	
" 12 ...	93-3	90-4	13 $\frac{3}{8}$ "	
" 13 ...	94-1	93-1	13 $\frac{3}{8}$ "	
" 14 ...	92	92-3	13 $\frac{1}{4}$ "	

Date.	Right knee.	Left knee.	Measurement of right knee.	Number of attack.
January 17, 18.	91.	91.		
" 19 ...	90.	90.		
" 20 ...	89.	86.		
" 21 ...	93.	91.5		
" 22 ...	93.	90.3	(Before attack.)	IX.
" 23 ...	95.	92.2		
" 24 ...	96.	95.		
" 25 ...	96.4	92.3	Catarrh.	
" 26 ...	97.1	94.3	—	
" 27 ...	95.8	94.2	—	
" 28 ...	94.2	94.3		
" 30 ...	93.2	92.4		
" 31 ...	90.3	89.2		
February 1 ...	95.2	94.1		
" 2 ...	97.1	97.1		
February 3 ...	94.	90.	.....	X.
" 4 ...	97.2	95.		
" 5 ...	95.	89.		
" 6 ...	96.	91.2		
" 7 ...	94.2	89.1		
" 8 ...	95.2	92.		
" 9 ...	89.	85.		
" 15 ...	95.4	95.4		
" 16 {	95.4	95.	.....	XI.
" 17 ...	96.3	93.		
" 18 ...	98.	96.		
" 20 ...	96.	95.		
" 21 ...	93.	93.		
" 22 ...	...	...	Catarrh.	
" 23 ...	91.2	90.		
" 24 ...	94.1	93.2		
" 25 ...	95.	92.3		
" 26 ...	95.2	93.		
" 27 ...	96.2	95.	.....	XII.
" 28 ...	96.2	94.3		
March 1 .....	97.3	96.2		
" 8 .....	...	...	.....	XIII.
" 9 .....	...	...	Catarrh.	
" 19 .....	...	...	.....	XIV.
" 31 .....	...	...	.....	XV.
April 7 .....	...	...	Catarrh.	
May 7 .....	...	...	.....	XVI.
June 12 .....	...	...	.....	XVII.
" 25 .....	...	...	.....	XVIII.
July 12 .....	...	...	.....	XIX.
" 26 .....	...	...	.....	XX.
August 6 .....	...	...	.....	XXI.

*Remarks.*—Although there was in each of these two cases a movable body in the knee, and inflammations may arise from over exercise in joints where such bodies exist, I cannot bring myself to believe that the phenomena I have described could be so accounted for. The precision with which washing days and ironing days return in some families might explain much, but not all this; and particularly such active family duties could not account for the punctual recurrence of the ailments, while the patients were in bed in the hospital, and when the limb of one of them was motionless in a splint.

In the first instance these singular transitory inflammations appear to have been due to some ordinary injury or local chill, but they were afterwards brought on afresh without a repetition of the original cause. The occasion of each periodic renewal of the ailment lay in the system of the patient, and not in the nature of the external injury. In the case of the elder woman, there could be no doubt that this tendency to repetition of a disease usually terminating at convalescence was a relic of the ague from which she had suffered in early life. It is true that the affection of the joint did not obey the precise law of the ague in respect to the duration of the successive periods; but neither was the ague itself quite accurate in its time rate, inasmuch as it was at one time quartan, at another tertian, and again at another quotidian. But, notwithstanding minor dissimilarities, the alternate outbreak and cessation of the symptoms over and over again was too obvious a character of both diseases to permit of any doubt of their essential connection with one another. The younger patient never had ague, and this proof of the nature of her disease was therefore wanting. But its resemblance to the former case, and its submission to the action of quinine, may be reasonably held to show that it was of the same nature.

My second case is not the only example of a periodic local disease arising without previous ague. Dr. Joseph Adams also was unable to connect the periodical vomiting with that cause in his cases; but conjecturing that they were of the nature of ague he treated them with arsenic successfully.

Certain neuralgias and other strictly local intermittents, taken together with the case of my second patient, appear to show that a periodical disease may be of the nature of ague, though it do not implicate the whole system, as agues usually do; that there may in fact be a primary ague limited to one part of the body, as well as an ague which is general.

If this be so, it must be erroneous to think of ague as an original entity, capable of entailing upon a future local disease the power of spontaneously reproducing itself at intervals. The transient characters of chill, heat, and liquid effusion, produced at first by a local injury, are indeed like those of an ague; but their recurrence over and over again is not due to ague, which may never have happened in the case, but to some more general fault in the system which occasions either separately, or is common cause to both.

The cases further appear to indicate that some local disorder is an essential part of the whole group of intermittents. For since a general ague may subside into a local periodical disease, and such local intermittent may, moreover, arise without any other sign of a present or preceding ague, the two differ only in degree, and there must be inferred to be at the root of the true ague, equally with the local intermittent, an obscure, but real, limited disease.

A main difference between the general and the local intermittent probably consists in the seat of this primary disorder. Pain only, or a limited inflammation, occurs where ganglionic nerves are few; but ague spreads over all the sympathetic system, from an organ endowed with none other. Even in a limb an acute abscess is often sufficient to evoke all the phenomena, except periodicity, of ague fits; but they are usually much more severe, more prolonged, and frequent, when the abscess is in an internal organ. And so an ague, limited to an external part, is slight and tardy, though still, like many nerve affections, periodic: but, when associated, as it notoriously rather is, with engorgement of the viscera, and especially of the spleen, it is distinguished for its severity, frequency, and comprehensive implication of all the system.

In speaking of these intermittent phenomena it is difficult to know whether they should be conceived of as causes or as effects, for the mechanism of the periodicity is hidden. But the adoption of a foregoing periodic character by a new and local morbid process, such as of itself usually terminates in convalescence, appears to show the affected part to be not primarily in fault. My first case makes it clear that the reappearance of an inflammation of the knee once over and gone was due to an unnatural state of the system introduced at the time of the long prior ague, with which the knee had nothing to do. The periodicity of the new disease was in fact provided for before it came on.

I have spoken of a fault in the system which may be supposed to give the character of periodicity either to an ague fit, or a local disease, or to both. It is a fault which I should suppose to reside in the nervous system, and especially among the ganglia. The recurrence of an ague or of a local intermittent may then be but the rebound of its own impact on the nerves of the disordered part; and this rebound may be due to a tendency of nervous influence, natural enough when not exaggerated, alternately to advance in its motion and to recede, oscillating in waves, of which the length and height are determined by conditions of the moving impulses. The nerve pendulum of an ague may thus swing to the measure of three days, or of one, from a force which in the nerves of the same patient's right knee-joint takes thirty days to recoil, or nine.

ON THE NATURE  
OF THE  
WAXY, LARDACEOUS, OR AMYLOID  
DEPOSIT.

BY  
WILLIAM H. DICKINSON, M.D. CANTAB., F.R.C.P.,  
ASSISTANT-PHYSICIAN TO ST. GEORGE'S HOSPITAL AND TO THE HOSPITAL  
FOR SICK CHILDREN.

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THIS disease, which was little noticed when described as waxy, lardaceous, or scrofulous, has of late years received much attention under the term "amyloid." It is not necessary to repeat what is already known with regard to the nature of the change. It is not limited to one organ; but many may become thus affected at the same time or in quick succession. A morbid deposit first appears in the walls of the small arteries, then penetrates their coats, and subsequently infiltrates the neighbouring tissues.

The small arteries, the walls of which are the seat of this deposition, acquire a peculiar reaction with iodine, a test by which they can be at once distinguished. Instead of the yellow colour which solutions of iodine give to all the healthy tissues, a deep reddish brown is produced by such solutions upon the affected vessels. It has been stated by Virchow



that, by a dexterous adjustment of sulphuric acid and iodine, a blue tint may be given to the "amyloid" deposit, but, like many other observers, I have never succeeded in obtaining any colour but reddish brown, merging into shades of dirty black. This colour, due to the precipitation of the iodine by the acid, would probably never have been looked upon as blue except by a person whose impartiality of observation had been warped by a desire to connect the morbid change with the production of starch.

Besides the change in the small arteries, which is the primary alteration, the several organs are affected, each in a manner peculiar to itself, by the exudation which the diseased vessels pour out. The solid viscera, such as the liver, spleen, and kidney, are increased in bulk, and, as best seen in the spleen and liver, become infiltrated with a new material which renders them firm, grey, and semi-transparent, characters which have suggested the term "*waxy*" as descriptive of the disease. A similar action taking place in the mucous membranes does not cause increase of bulk, but the exudation seems to pass, as a secretion, from the free surface of the membrane thus affected.

The new deposit, as may be clearly seen in sections of the kidney, is found to lie in abundance around the vessels from which it has exuded, and to permeate more or less the whole structure of the gland. This new formation readily becomes converted into fibrous tissue. Where seen in bulk it soon assumes, under the microscope, a fibro-nucleated structure, and its presence in small amount is evinced by a thickening of all the fibrous structures with which it comes in contact. After its deposition it undergoes a regular process of contraction, as is evinced by the changes which take place in the parts involved. The new material occasionally gives the same reactions as the affected vessels. In some cases the fibrinous casts, which lie in numbers in the tubes, give the reaction in a very striking manner (see Plate I). The matter which infiltrates the gland appears to be identical in all respects with that which forms the casts; the only difference is in the position of the vessels from which the

exudation takes place. The vessels which are imbedded in the tissue are the source of one exudation, while the exposed Malpighian tufts supply the other: the matter from one source runs into the tissue, from the other into the tubes. Whether it appear in one place or the other it is evident that the exudation is essentially the same.

Organs affected by this disease have been repeatedly subjected to ultimate analysis. I may here refer to the valuable researches<sup>1</sup> of Drs. Pavy and Odling. The results prove, beyond question, that the addition to their substance is either albuminous or fibrinous. The facts which have been stated regarding the new growth, its convertibility into fibrous tissue, its contractile tendency, and its apparent identity with fibrinous casts, appear to prove that it is essentially fibrinous.

It is my purpose at present to consider the disease in its general relations, rather than as affecting any organ in particular.

It is evident from the number of organs affected at the same time that the "amyloid" deposit is the result of an agency ubiquitous and simultaneous through the system. It is known that the change is apt to occur after exhausting diseases, particularly such as are syphilitic or scrofulous in their nature, and such as concern bone. Dr. Wilks<sup>2</sup> associates the disease with "long-standing and deep-seated cachexia," such as proceeds from tubercular or syphilitic disease, particularly if it affect bone. Dr. Grainger Stewart, who has collected many cases, associates the disorder with scrofula, caries, necrosis, and syphilis, or with feebleness of constitution without palpable disease.

I hope to be able to show that, whatever the nature of the antecedent disease may be, there is one definite process, common to many disorders, which is the great cause of the morbid deposit.

I have been able to collect from my own experience, in the dead-house and elsewhere, the particulars of sixty cases of

<sup>1</sup> 'Guy's Hospital Reports,' 1864.

<sup>2</sup> 'Guy's Hospital Reports,' 1865.

this disease. I can answer for the state of the organs after death in the whole number. The circumstances of the case during life have in some instances come under my own knowledge, while in others I have obtained a more or less complete history from the St. George's Hospital records, and other sources. The following table gives the nature of the disorder which, in each case, was succeeded by the waxy or "amyloid" infiltration.

TABLE I.

*Analysis of sixty cases of so-called "amyloid" or "waxy" degeneration, which have come under the observation of the author, showing the affections by which the change has been preceded.*

Direct evidence of suppuration.	Presumption of suppuration.	No evidence relating to suppuration.
Exposed bone from caries or necrosis, with long-continued purulent discharge ..... 18	Phthisis with extensive cicatrices ... 2 Disease of elbow-joint, for which arm was amputated ..... 1	Cause unknown... 5 Following diseases produced by tropical climate 1 Apparently, consequent upon albuminuria, "tubal nephritis"..... 4
Disease of spine, with psoas abscess ..... 1	Extensive disease of spine with much curvature and loss of bone ..... 1	
Profuse suppuration, connected with amputation of thigh... 1		
Suppuration consequent upon compound fracture of leg..... 1		
Phthisis with vomicae 9		
Dilated bronchi with profuse purulent expectoration ..... 1		
Ulcerated cancer ..... 5		
Ulceration of bowel, including dysentery ..... 6		
Destruction of kidney by tubercular excavation (1 case causing psoas abscess) . 2		
Severe syphilitic ulceration of throat or penis ..... 2		
Total..... 46	Total ..... 4	Total ..... 10

In fifty cases the change appeared to be associated with the formation of pus; in four with the loss of albumen by the urine. In the remaining six the source of the disease was not traced.

In further detail there were forty-six cases in which there was evidence of a profuse and long-continued drain of pus. In most of these the discharge persisted until the death of the patient. In four the post-mortem appearances were such as to lead to the inference that a discharge of pus had taken place at some remote period; in two there had been tubercular disease of the lungs with extensive cicatrices; in two, destruction of bone by disease of uncertain character.

Excepting in the concurrence of suppuration with the "amyloid" deposit these cases have no resemblance to each other. Syphilis had existed in some, tubercular deposit in others;<sup>1</sup> while in some there was no evidence of any constitutional disease.

Of the ten remaining cases, in which there was no evidence implying the loss of pus, there were four in which there was reason to believe that the "amyloid" change had followed upon albuminuria. The albuminuria, in each case, was of the kind which is associated with tubal nephritis, and which is characterised by a large discharge of albumen. In two of the cases the urine also contained pus, though in small quantity, which apparently had been secreted by the renal tubes.

In these four cases it may be mentioned that the "amyloid" change was in an early stage, in three of them confined to the kidney.

The practical and pathological importance of the conclusion that associates the "amyloid" deposit with the suppurative process has led me to search the recorded experience of other observers for evidence in confirmation or correction of this view.

Dr. Wilks has published two papers containing the par-

<sup>1</sup> It may be worth while to state that in these 60 cases there was a history of syphilis in 4, in each of which there was either extensive destruction of soft parts, or necrosis with prolonged suppuration. The cases in which cancer or tubercles were present are sufficiently indicated in the table.

ticulars of ninety-six cases of this disease. A statement of the causes to which he has traced the disorder is given in a table.

TABLE II.

*Analysis of ninety-six cases of waxy or lardaceous disease, showing the disorders by which it has been preceded. Cases published by Dr. Wilks, in 'Guy's Hospital Reports' for 1856 and 1865.*

Direct evidence of suppuration.	Presumption of suppuration.	No evidence relating to suppuration.
Purulent discharge indicated in connection with diseased bone ..... 24	Disease of bones or joints, nature not specified ..... 5	Cause not ascertained; chiefly from incompleteness of history ..... 11
Cases described as necrosis ..... 4	Syphilitic affection of bone ..... 7	
Chronic ulcers involving bone ..... 2	Syphilis; no description ..... 2	
Scrofulous sores ..... 3	General tuberculosis. .... 2	
Scrofulous disease of urinary organs..... 3	Tubercular peritonitis ..... 1	
Phthisis (lung generally described as disorganised) ..... 20		
Syphilitic ulceration of soft parts..... 6		
Dysentery or ulceration of bowels ..... 4		
Suppuration in pelvis after labour ..... 2		
Total..... 68	Total ..... 17	Total ..... 11

In eleven of the number, from the incompleteness of the history and other circumstances, the origin of the disease was not discovered. Of the remaining eighty-five there were sixty-eight in which there was direct evidence of a suppurative drain, while in seventeen the nature of the post-mortem appearances gave a fair presumption that suppuration had gone on at a former period.

I have further been able to collect from three papers published by Dr. Grainger Stewart the particulars of twenty-seven cases of the amyloid change in which the body was



Placing together the cases from both writers, we have 109 in which the antecedent disease was traced; of these 83, or more than three quarters, were consequent upon undoubted loss of pus, while in the remaining 26 the preceding disorders are of such a nature that it is not possible to doubt that the same morbid discharge must, at some period, have been present in the greater number.

If the existence of a protracted discharge of pus be ascertained in any case under treatment, even though there may be no symptom of "amyloid" disease, it may be surmised that some amount of the peculiar change will be found on post-mortem examination. And if in such a case there be albumen in the urine, or palpable enlargement of the liver, the existence of the "amyloid" deposit may be confidently assumed.

It is necessary to abandon the view that the disease in question is necessarily connected with any especial cachexia or specific disease.

Three of the cases which came under my own knowledge are instances in support of this remark.

A child, 6 years of age, was under my care at the Children's Hospital, for cough with purulent expectoration, which dated from an attack of whooping-cough four years before. Bubbling and cavernous sounds were heard in the lungs, and it was presumed that she had phthisis. While under treatment œdema appeared, and the urine was found to be albuminous. The existence of "amyloid" degeneration was inferred, and the treatment was modified accordingly. When, after death, the body was examined, the anticipated change was found in the liver, kidneys, and intestines. But the cause of the purulent expectoration was not phthisis. The lungs were full of cavities, some as large as walnuts, formed by dilatation of the bronchial tubes. There was not a tubercle in the body. The character of the expectoration in this case will be described subsequently. (See page 48.)

A life-guardsmen while in perfect health received a severe compound fracture of the leg, his horse falling upon him. The wound healed slowly, after having suppurated for about

two months. The man after a time recovered apparent health. Eventually, without any further cause, as far as could be ascertained, he was attacked with dropsy and albuminuria, of which he died nearly six years after the accident. The kidneys were preserved as specimens of the "large smooth kidney." Some years later, the concurrence of the renal disease with the injury suggested their true character, and on re-opening the preparation the iodine test showed them to be "amyloid" in a most marked degree.

The third case was that of a vigorous young man whose thigh was amputated in consequence of an accident by which the popliteal artery was torn. Most profuse suppuration followed the operation, and twenty-one days afterwards the patient died of pyæmia. The characteristic iodine-reaction was detected in the malpighian bodies of the kidneys.

Among Dr. Wilks's cases are two in which the disease followed suppuration in the pelvis consequent upon labour.

The relation which subsists between the subtraction of pus from the system, and the deposition of the new material in the organs, is the next step in the inquiry. It will be necessary to have regard to the composition of pus on the one hand, and of the so-called amyloid deposit on the other.

Pus is an albuminous fluid, which is alkaline, owing to the presence of a large amount of potass and soda. Earthy salts are also present, but in comparatively minute quantity. Fibrine is absent. It may be roughly estimated that the alkaline and earthy salts of pus amount to 1 per cent. of the discharge, and that of these the salts of potass and soda form about nine tenths. It will be shown afterwards that there are characters in the new deposit which draw attention in an especial manner to the alkalies. It is not necessary to repeat in this place details of analysis which are familiar to all pathologists. I have appended in a note some of the results which have been obtained by chemists.<sup>1</sup>

<sup>1</sup> *Note on the composition of pus.*—Three analyses of pus were made by Dr. Wright ('Med. Times,' 1845), the discharge having been obtained respectively from a vomica, a psoas abscess, and a mammary abscess. The salts of the alkalies and lime varied, in 1000 parts, from 8·9 to 13·5.

Analyses of pus are given in Simon's 'Chemistry' (Cavendish Society,



In the case of dilated bronchi, followed by "amyloid" change (page 46), the child coughed up daily a quantity of pus, which did not vary much from 105 cubic centimetres, or about three ounces and a half.

	Grms.
This contained	·922 of mineral matter.
	·867 of alkaline salts (about 13 grains).
	·055 of earthy salts.

The alkalies amounted to—

Soda	.	.	·275
Potass	.	.	·113

The above quantities represent the secretion of twenty-four hours.

In some pus obtained from a psoas abscess I found that 100 parts contained, of potass ·504, of soda ·476, the earthy salts, not analysed, amounting to ·2.

From these particulars it appears, that not only does pus contain alkaline salts in the amount stated, but that these salts are much more abundant in pus than in the blood from which the secretion is derived. A discharge of pus is equivalent to a removal from the blood of albumen and alkali. Having regard to the great length of time for which the drain is sometimes continued, and the large daily amount

vol. 2, p. 91), in which the proportion of the soluble salts of pus to the insoluble is estimated in one case as 5 to 0·7, in another as 4·7 to 0·62.

The analyses made by Nasse of the serum of pus are compared with the serum of blood; the general results are as follow: In 1000 parts of the serum of blood the alkaline salts amounted to 7·1. In the same quantity of pus serum the alkaline salts amounted to 15·32, more than twice as much.

Lehman ('Physiological Chemistry,' Cavendish Society, vol. 2, p. 150) made comparisons between the composition of pus and of blood taken from the same animal, conducting his experiments upon wounded rabbits and geese. With rabbits he shows that while the ash of pus contains 6·9 per cent. of potass, the ash of the corresponding blood contains only 4·8 per cent. With geese the difference is still greater. This chemist infers that "the ash of pus always contains a larger amount of phosphates and potass salts than the inter-cellular fluid of the corresponding blood."

which may be discharged, we may be prepared to expect consequent changes in the nutrition of the body.

What is true of a purulent discharge holds good in a modified degree with some forms of albuminuria. In albuminuria there is often a loss both of albumen and free alkali. It does not appear that the *salts* of the alkalies are more abundant in albuminous than in healthy urine; but albuminous urine is generally wanting in acidity, sometimes positively alkaline. Whether this arises from excess of alkali or deficiency of acid the same result must follow—a diminution of the free alkali of the blood.

I may now take into consideration the characters of the “amyloid” or “waxy” deposit. It is no longer necessary to discuss the theory which associates it with the development of starch. It has been already shown that the results of ultimate analysis, the contractile tendency of the new formation, its transformation into fibrous tissue, and its apparent identity with fibrinous casts, concur to prove that it is essentially fibrinous. But since fibrine does not, in a general way, give the characteristic reaction with iodine, it remains to be shown on what this reaction depends. In order to do so I must ask the attention of the Society to some chemical details.

The deep brown colour with iodine, which is characteristic of the “amyloid” disease, is constant under a variety of circumstances. The affected tissue may be dried, boiled, soaked in alcohol, in strong acids, or in caustic ammonia, and still the same striking colour is brought out by the same reagent. It is not affected by solutions of any of the alkaline salts; but if a section of an organ so diseased be allowed to remain for a short time in a dilute solution of caustic potass or soda, the colour soon disappears never to return. The destruction of the iodine reaction is permanent, although all removeable alkali be washed from the tissue; the reaction is not restored by the action of acid. The action of iodine becomes precisely what it is upon healthy structure. The minute quantity of potass or soda which the tissue takes up destroys the so-called amyloid reaction. The power of destroying the

reaction is, as far as I can ascertain, confined to caustic potass and soda: it does not extend to their salts, or to ammonia in any form.

After many experiments upon "amyloid" tissues, I learned that sulphate of indigo is a test for the deposit no less striking than iodine itself. If a piece of a healthy organ be soaked in a weak solution of this material it assumes a blue colour, which, after a time, fades into a pale greenish tint (Plate II, Fig. a). This change also takes place in the parts of an "amyloid" organ which escape the disease. Those portions, however, which are affected retain a deep brilliant blue which affords a striking contrast with the parts which are free from the morbid deposit. Thus it appears that the healthy tissue of the liver or kidney has a power of destroying the colour of sulphate of indigo, which the morbid deposit does not possess. The power of decolourising sulphate of indigo is known to belong to the caustic alkalis; under the circumstances described the destruction of colour appears to be due to the free alkali contained in the healthy tissues. The colour is retained in the morbid deposit, and in that only, because it is the only part without this component. The drawings (Plate II, Figs. b and c) show the effect described.

I might adduce other arguments to show that the morbid deposit differs from healthy tissue in the absence from it of free alkali (the action of turmeric is characteristic); but it is not necessary to multiply observations in behalf of a conclusion which will be placed beyond doubt by the remaining steps of this inquiry.

I will now proceed to give the results of some analyses of "amyloid" as compared with healthy organs, undertaken with a view of ascertaining the proportion of potass and soda in each. The liver has been made use of for this purpose, because it contains a greater apparent amount of the morbid formation than can be found elsewhere. But even in this structure it must be borne in mind, that the deposit is interspersed through the normal structure of the gland, to which in most cases it bears but a small proportion.

TABLE IV.

*Table showing per-centage of alkaline and earthy salts in seven "amyloid" livers.*

Case.	Soluble Ash.	Insoluble Ash.	Potash.	Soda.	Potash and Soda together.
Mary Morgan. Large bed-sores; liver in very early stage; kidneys more affected .....	1·075	·062	·203	·177	·38
Robert Nutt. Large vomica; liver slightly affected, kidney highly .....	·96	·098	·256	·154	·410
Sent from Edinburgh. Highly affected; somewhat dry when examined .....	·852	·21	·189	·206	·395
From Consumption Hospital. Highly affected; large vomica .....	·677	·245	—	—	—
Thomas Theakstone. Tubercular ulceration of bowels, &c.; decidedly affected .....	·621	·18	·163	·153	·316
Benjamin Thomas. Disease of pelvis, protracted discharge; intermixed with fatty change .....	·614	·262	·086	·19	·276
John King. Profuse suppuration from diseased wrist, phthisis; decidedly affected; kidneys also .....	·481	·209	·12	·053	·173

Average per-centages—Soluble ash, or total alkaline salts, ·754; potash, ·169; soda, ·156.

*Per-centage of alkaline and earthy salts in seven healthy livers.*

Case.	Soluble Ash.	Insoluble Ash.	Potash.	Soda.	Potash and Soda together.
Liver of an adult; tissue apparently natural .....	1·181	·053	·187	·255	·442
Ditto .....	1·11	·173	·23	·189	·419
Liver of an adult; tissue slightly congested .....	1·041	·033	·205	·252	·457
Liver of an adult; tissue apparently natural .....	·995	·016	·214	·192	·406
Ditto .....	·912	·037	·22	·25	·47
Ditto .....	·904	·05	·191	·123	·314
Ditto .....	·898	·041	·215	·164	·379

Average per-centages—Soluble ash, or total alkaline salts, 1·00; potash, ·209; soda, ·196.

It is not necessary to occupy the Society with details of analysis. It may be shortly stated that a certain weight of the organ, as removed from the body, was reduced to an ash, and the saline constituents extracted in the usual manner. In some cases the acids were estimated as well as the bases, but at present it is only necessary to pay attention to the amount of alkali.

I have estimated the potass and soda in seven livers at different stages of the disease, and I have, for the sake of comparison, examined in the same way seven healthy livers: the results are stated in a tabular form. The analyses, to facilitate comparison, are arranged according to the amount of alkaline salts.

It will be seen that the proportion of alkaline salts in the healthy organ does not undergo much variation, while in the "amyloid" it varies greatly in consequence of its irregular diminution. In the healthy specimens the alkaline salts varied between 89 and 118 parts in 1000. With the amyloid the variation was between 48 and 107. Taking the amounts of potass and soda separately a similar statement will hold good. In the diseased state both are irregularly diminished. The amount of reduction will be best estimated by comparing, as regards each item, the average of health with the average of disease.

Taking the gross alkaline salts in 100 parts of the fresh liver, it appears that the average of health is exactly 1·00; the average of "amyloid," ·754, a reduction of one quarter. Taking the alkalies by themselves the healthy average of potash is ·209; the "amyloid" average, ·169, a loss of one fifth. The healthy average of soda is ·196, the "amyloid" average, ·156, a loss also of one fifth.

Hence it appears that a given weight of the "amyloid" liver contains less potash and less soda than the same weight of the healthy liver by about one fifth. The alkalies continue to hold about their natural healthy proportion to each other.

It is worthy of remark that while the alkalies of the "amyloid" organ are diminished the earthy salts are increased.

From what has been brought forward it seems that the reactions are due to the absence of alkali, while the formation is essentially fibrinous. Under these circumstances we ought to be able, by depriving fibrine of the alkali with which it is ordinarily combined, to manufacture a material which has the "amyloid" reaction; and this can be done.

Fibrine is soluble in dilute hydrochloric acid (0·6 in 1000). If the solution be evaporated to dryness at a low temperature, a substance is obtained which has all the reactions characteristic of "amyloid" disease. The acid has necessarily deprived the fibrine of its alkali.

Ordinary fibrine comports itself with iodine and sulphate of indigo, as do the healthy tissues. It takes a yellow colour with iodine; indigo gives it a blue colour, which fades, and eventually is destroyed. Fibrine, which has been *dealkalised* by the process described, reacts precisely as does the morbid deposit. Iodine gives to it the rich red-brown, while the bright blue of indigo is retained for an indefinite time.

The same reactions are afforded by albumen which has been deprived of alkali by a similar method. The reactions are the same, though all free acid has been neutralised by carbonate of ammonia, showing that they are due to the absence of fixed alkali, not to the presence of the acid.

If it be wished to exhibit the characteristic reactions of "amyloid" deposit the artificial product answers the purpose extremely well, in consequence of the marked character of the colours produced.

**Conclusions.**—The so-called "amyloid" or "waxy" deposit consists of dealkalised fibrine. It is not necessary to repeat the facts which lead to the conclusion that the deposit is fibrinous. That it is wanting in alkali is shown by these considerations:

1. The morbid deposit loses its characteristic reactions when it has been allowed to absorb potass or soda.

2. Organs containing this deposit yield on analysis a smaller proportion of the alkalies than do the same organs in a state of health.

3. Ordinary fibrine or albumen can be made to exhibit all

the peculiarities of "amyloid" tissue by depriving them of alkali by artificial means.

The morbid deposit is of the nature of a residuum; it occurs in cases where the system has been drained by an alkaline and albuminous discharge, the blood, therefore, containing an excess of fibrine with a deficiency of potass and soda. Possibly the removal of the alkali may occasion the deposition of the fibrine.

The most frequent cause by which this deposit is produced is suppuration, a cause which is active in at least five cases out of six. The loss of albumen by the urine has a feeble tendency of the same sort. What other causes produce the disorder, and whether there are conditions which render the system more than usually liable to be affected by such processes as have been designated, are questions as yet unanswered.

The frequency of "amyloid" disease, the obvious nature of its cause, and the readiness with which it can be detected during life, combine to give it great practical importance. It supplies a large proportion of the albuminuria which comes under the care of the physician, and it is of constant occurrence among surgical patients, modifying the course of disease and overruling the results of operations.

The term *amyloid* must fall into disuse as founded upon error. The terms *waxy* and *lardaceous* are applicable to the change produced in a few organs only, besides which, like most pathological adjectives derived from superficial and often imaginary resemblances, they have each been applied to more than one sort of morbid formation.

I venture to propose the word *Depurative*<sup>1</sup> as significant of the process which is the most frequent source of the disease. Under this designation the false conceptions would be avoided which attach to the phrases by which the morbid change has hitherto been described, and a clear assertion would be made

<sup>1</sup> It has been objected to this word that depurate is already in use in the sense of purify. This, however, appears to be of small consequence, since no confusion is likely to result. The fault rests with those who originally committed the identity of root between the words *pus* and *purus*.

of the great practical truth that in all cases of protracted suppuration the deposit in question is a probable contingency.

The inquiry is not barren of a suggestion with regard to the medical treatment of a purulent discharge. Our endeavour must be to compensate for the loss sustained by the system. The diet may be regulated so as to include abundance of albuminous matter, while the alkalies and their vegetable salts may be given in due proportion, and according to the circumstances of the individual case.

In conclusion, I have to thank Dr. Wilks, Dr. Grainger Stewart, Dr. R. Douglas Powell, and Dr. G. C. Bright, who have provided me with specimens for examination; also Dr. Noad, who has given me much valuable assistance in the chemical part of the investigation.



### DESCRIPTION OF PLATE I.

Amyloid kidney, showing casts *in situ*, coloured by iodine in the same manner as the Malpighian bodies and small arteries.  $\times 75$  diameters.

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### DESCRIPTION OF PLATE II.

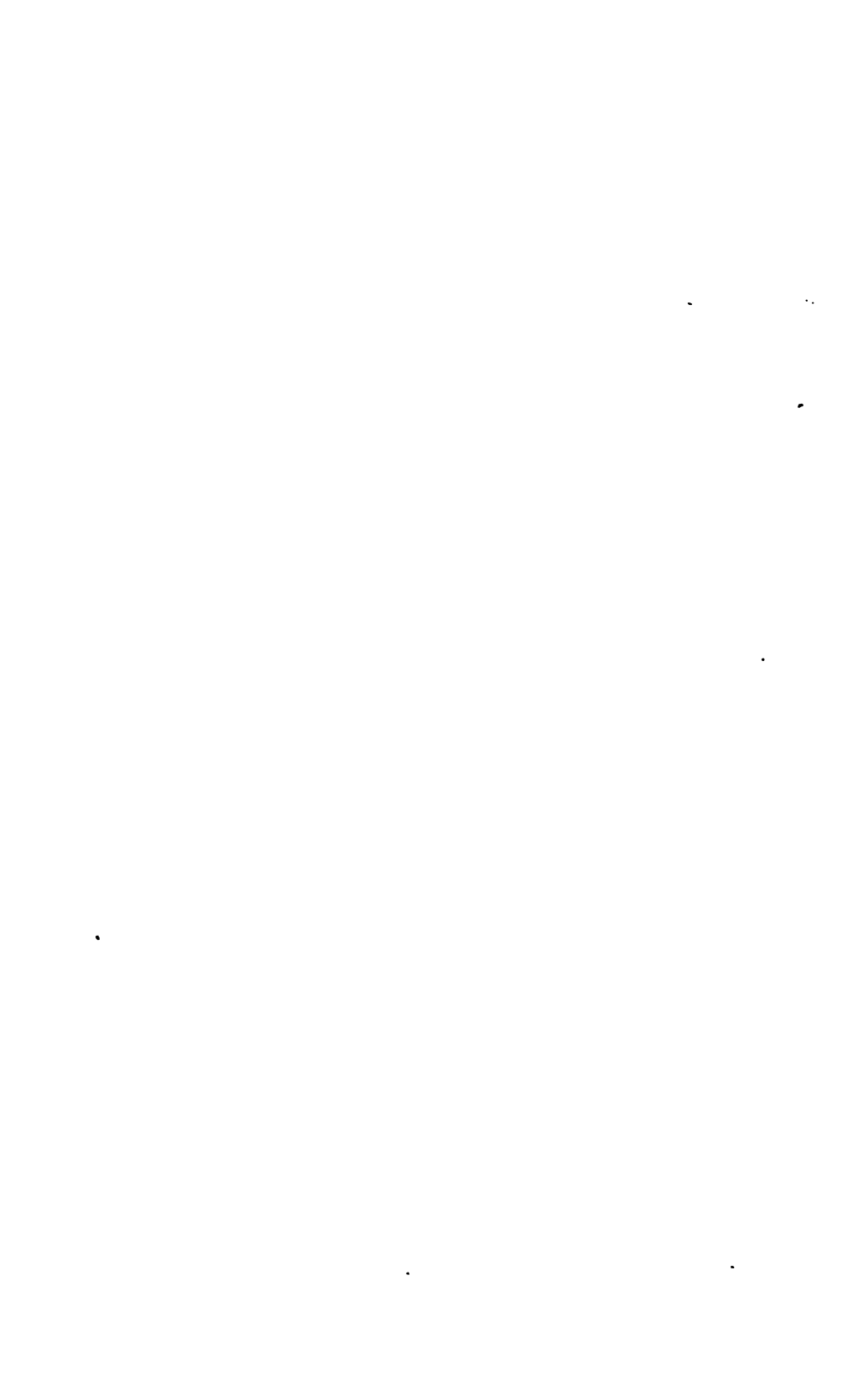
FIG. A  $\times 50$  diameters. Section of a healthy kidney, coloured with indigo, showing the uniform fading.

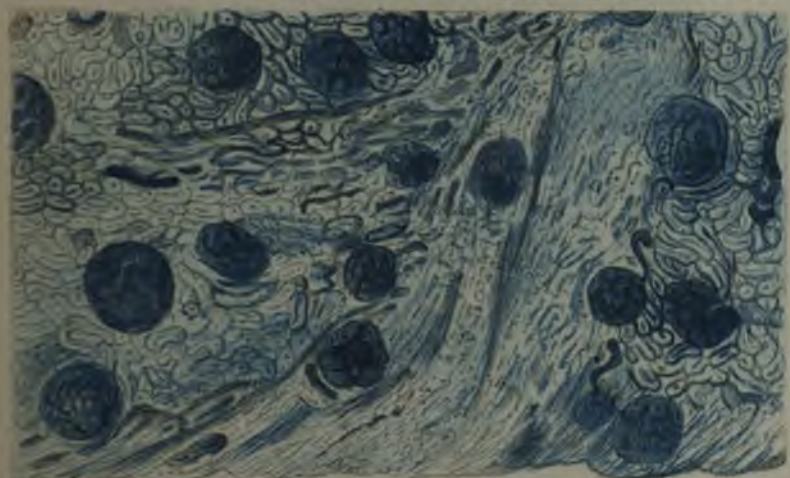
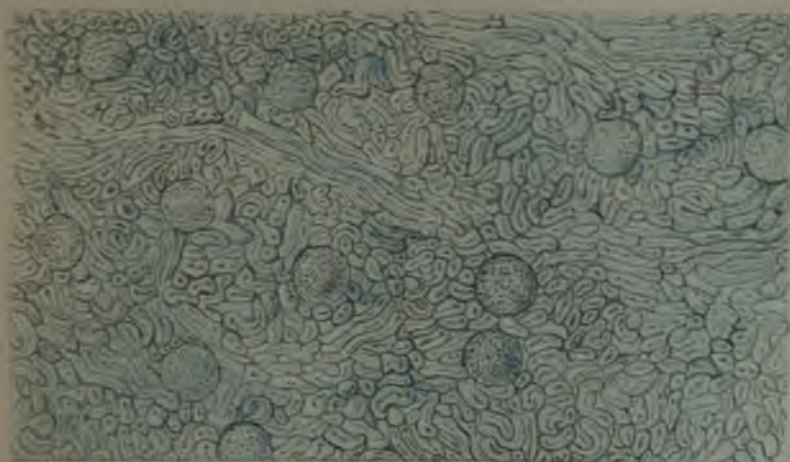
FIG. B  $\times 50$  diameters. Amyloid kidney; cortex, including capsular edge; coloured with indigo, showing the retention of colour on the affected parts, the Malpighian bodies.

FIG. C  $\times 50$  diameters. "Amyloid" kidney coloured with indigo (medullary part), showing the retention of colour upon the affected vessels.

N.B. The three sections coloured with indigo were kept for the same time in the same solution.









ON  
ENUCLEATION OF NÆVUS.

BY  
T. PRIDGIN TEALE, JUN., M.A. OXON., F.R.C.S.

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IN the following paper I propose to advocate, and illustrate by cases and specimens, a method of dealing with large subcutaneous nævus, which appears to me to have received less attention, in operative surgery, than it deserves. Having for several years adopted more and more exclusively the method of extirpation by the knife, in preference to the various modes of producing destruction or atrophy of the nævoid structure by ligature, setons, nitrate of silver, or injection, I have been forcibly struck with the degree in which in many cases the nævoid growth was distinct from the neighbouring tissues. The conclusion was gradually forced upon me that in a large majority of cases in which the nævus was more or less subcutaneous, the new growth was surrounded by condensed connective tissues forming a more or less perfect capsule, and that this limiting capsule was sufficiently distinct to enable the surgeon to coast along the surface of the tumour, enucleating to a great extent, and using the knife here and there if necessary to assist the process of enucleation.

Over the centre of the tumour there was the scar resulting from the use of the ligature nine weeks previously. The tumour was soft and lobulated, and after being partially emptied by pressure, slowly refilled like a sponge.

Having explained my views as to the existence of a capsule and the possibility of enucleation, I obtained the approval of the hospital staff to the course I proposed to adopt, and with the assistance of Mr. S. Smith, my father, and others, carried out the operation in the following manner.

Having isolated the scar resulting from the previous insertion of the ligature, I reflected in flaps the skin covering the surface of the nævus, including in the flaps the nævoid as well as the sound skin. The outer surface of the mass having been laid bare, mainly by careful dissection by the knife, the operation was completed by tearing and enucleating the tumour from its deep-seated connections with comparatively little use of the knife. During the early part of the operation the child was kept under the influence of chloroform, and towards the latter end became faint, vomited, and then rallied. The chief hæmorrhage occurred during the reflection of the skin, but was controlled by the fingers of those assisting me. On separating the deep surface there was comparatively little bleeding. Only five vessels required ligature. About half an inch of the internal jugular vein was exposed during the operation, and the styloid process could be felt by the finger. A small portion passing deeply in front of the styloid process was ligatured, as it was not thought safe to risk the sudden hæmorrhage that might occur from one of the larger branches of the external carotid.

I was not able to make out whether a portion of parotid gland had been removed along with the nævus; or whether the diseased growth had originated in the parotid and had extended outwards until it reached the skin. The infant rallied quickly and recovered without any unfavorable symptom.

The history of the diseased portion of skin is interesting. About one third sloughed. The remainder became gradually

drawn down over the wound by cicatrization, and at the end of three months had the usual appearance of cutaneous nævus, being as large as a shilling. At the end of a year all appearance of nævus had faded away.

The tumour was composed of a lobulated pinkish mass, with large venous sinuses in the middle limited by walls of very slender structure hardly to be distinguished from the investing membrane of the lobes of the tumour. A hasty microscopical examination did not reveal any distinct structure.

The operation was followed by facial palsy of the right side, which at the end of two years had diminished, but had not completely ceased.

CASE 2.—Alice B—, æt. 7 months, a patient of Mr. Edwin Moore of Halifax, was admitted into the Leeds Infirmary, in January, 1865, on account of a large nævus situated over the left parotid gland. The tumour was first noticed a month after birth as a bluish mark under the left ear. A month later it became more blue and swollen, and was punctured to ascertain whether it was an abscess or not. The puncture was attended by considerable flow of blood. The tumour had doubled in size during the last three months, the growth being most rapid during the last month. It measured 4 inches by  $3\frac{1}{4}$ , and was entirely subcutaneous, and could be much reduced in size by pressure.

January 19th, 1865.—Chloroform having been administered, the skin was carefully reflected from the surface of the tumour, and the operation was then rapidly completed by enucleating the mass from its bed, partly by the handle of the scalpel and Key's director, and partly by tearing it away by the fingers.

During the early part of the operation there was considerable hæmorrhage, the vessels passing from the tumour to the skin being numerous. From the deep surface of the wound the bleeding was slight. Five ligatures were required.

The next day the child was faint, and had two convulsive fits. After the first two or three days its condition rapidly



improved, and it was able to return home to Halifax in ten days, convalescent.

A few days after returning home it was seized with malignant scarlet fever and died; the wound being reported as healthy, and healing well before the fever set in.

CASE 3.—Although the nævus in this case was not large, and was much less formidable than the two preceding ones, I publish it along with the rest, as the fatal result must be attributed directly to the operation.

Mary R—, æt. 5 months, a patient in the Leeds Infirmary, admitted on March 31st, 1864. A nævus, chiefly subcutaneous, of the size of a walnut, was removed from the right parotid region by enucleation aided by the knife. The deep surface was easily separated without the use of the knife, and the capsule covering the parotid was recognised at the bottom of the wound. The wound became swollen and unhealthy, and the child died on the fifth day, in a fit of laryngismus to which it had been subject. In this case there was no facial palsy after the operation.

On reviewing the foregoing cases it may be remarked, 1st, That all the patients were of tender age, the youngest being four months old, the eldest seven months; 2nd, That in two the disease was advancing with great rapidity; 3rd, That in all, the existence of a capsule and the possibility of enucleation was established; 4th, That in the first case, after the lapse of twelve months, the preserved nævoid skin had lost its undue vascularity.

*Note on the danger of injection of chemical fluids into nævus.*

About six years ago an infant, æt. 6 weeks, came under my care on account of a large nævus situated at the edge of the right pectoral muscle, which was increasing rapidly and had attained the size of a billiard ball. As the treatment by injection by perchloride of iron was then on its trial and had succeeded in several cases which I had seen, I determined to adopt it in this case.

Several punctures were made and a few minims of the perchloride were introduced at each puncture. The greater part of the tumour became rapidly solidified. Three weeks later the effect of the injection had passed off and the tumour again began to increase rapidly. I therefore repeated the injection. After three or four punctures and injection of the tincture of iron the child looked pale, became convulsed, and died in a few minutes.

At that time I supposed that the convulsion was nothing more than such as is occasionally set up in infants by comparatively slight causes. I now feel convinced that the needle had penetrated a large venous sinus in the centre of the tumour, and that blood coagulated by the perchloride had been carried to the heart and had there arrested the circulation. Unfortunately I was unable to make a post-mortem examination.

P.S.—The anatomical characters of subcutaneous nævus, and especially the existence of a capsule, are well described by Mr. Birkett in the 'Medico-Chirurgical Transactions' for the year 1847. Mr. Birkett also infers, as a result of his examination of the tumour, the danger of treating nævus by injection of chemical fluids.



ON A CASE  
OF  
INTERNAL STRANGULATION OF THE  
BOWEL BY A BAND,  
ASSOCIATED WITH A REDUCIBLE HERNIA;  
SUCCESSFULLY TREATED BY OPERATION.

WITH REMARKS.

BY  
THOMAS BRYANT, F.R.C.S.,  
ASSISTANT-SURGEON TO GUY'S HOSPITAL.

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On December 31st, 1866, I was asked by Dr. Wilkinson, of Sydenham, to see with him a gentleman, æt. 51, who had been ill for several days with symptoms of intestinal obstruction. The patient, Mr. H—, had been the subject of an inguinal hernia on the *right* side for twenty-five years, for which he had worn a truss; during that period the bowel had come down on several occasions, but it had only given trouble on one—some six months previously.

On the morning of December 28th, during the exertion of dragging up a tree, the hernia partially descended, but it was at once readily returned on the application of the hand; vomiting, however, soon appeared, and pain situated on the

right side of the umbilicus. These symptoms continued on the 29th and 30th, and increasing in severity, Dr. Wilkinson was sent for. I may here add that Mr. H—'s previous health had been good, and his bowels regular. In infancy he had disease of the left hip-joint, which had become ankylosed.

On December 30th a careful examination was made, but no hernia was found; there was a large opening into the abdomen, in the region of the inguinal canal, through which the extremity of the finger could be passed, but no swelling was present, and not even pain on deep pressure being made. The cause of the symptoms was evidently obscure.

On December 31st, or the third day, the symptoms remained unchanged in character, and becoming more severe—the vomiting being fecal and constipation present—Dr. Wilkinson, who saw the immediate necessity for an operation, requested my assistance.

When I saw the patient at 2 p.m. I found him in bed, with a somewhat anxious countenance. He had recently vomited fecal matter and was constantly retching. He complained of a fixed pain on the right side of the umbilicus, which became at times worse and paroxysmal. The abdomen was evidently abnormally distended, but it was not tense. The seat of the old hernia on the right side was carefully examined, and the ring through which the hernia had descended made out, but nothing like a swelling, or even pain on pressure on the part, could be detected. The groins were also carefully examined for a femoral or even an obturator hernia, but without result. The patient was made to stand, in the hope that under such circumstances some slight evidence of a hernial swelling might become manifested, but without effect, no trace of any such condition making its appearance.

From the fecal vomiting and constipation there was no room for doubt that intestinal obstruction existed; from the absence of any fulness or pain even on pressure in the seat of the old hernial swelling, and in the femoral and inguinal regions, it was difficult to believe that the symptoms were due to any ordinary hernia; one thing only seemed clear, that death was

imminent if relief was not given; under these circumstances I deemed it right to suggest the propriety of an exploratory operation, and in this suggestion Dr. Wilkinson cordially coincided. The difficulties of the case were then explained to our patient, who was unusually intelligent, and his assent readily secured.

The seat of the old hernia was naturally selected for the exploratory incision, and more particularly as it was a few inches above this spot that the fixed abdominal pain had located itself. Dr. Wilkinson and Mr. Giles kindly gave every assistance; chloroform was given by Dr. Wilkinson, and during its administration some retching took place; it was then thought that a slight bulging became visible in the seat of the old hernia, but this was not very marked.

The incision was made in the right groin over the inguinal canal, and the ring of the old hernia carefully exposed; the sac of the hernia was opened, and a small piece of adherent omentum found. The abdominal opening through which the hernia had descended was then clearly made out to be on the inner side of the inguinal canal, proving that the hernia had been a direct one; it was as large as a sixpence; through this and within the abdominal cavity some small intestine was then seen, of a bright cherry colour; to the touch this felt thickened, and was evidently œdematous. All these parts were then carefully explored; the neck of the hernial sac was drawn down, and the finger introduced through the hernial opening into the abdominal cavity, but nothing abnormal could be found, and no evidence of strangulation of the bowel by any of the parts which had entered into the formation of the hernia made out. The finger could be moved readily in every direction within the abdomen. Under these circumstances the coil of intestine, which was injected and œdematous, was carefully withdrawn through the abdominal opening, which had been previously enlarged, and dragged gently downwards; the index-finger of the right hand was passed along its lower border into the abdomen towards the spot at which the patient had complained of the fixed pain, near the umbilicus, and when it had passed completely out of sight, and as much traction

had been made upon the bowel as was deemed prudent, a tight, tense, fibrous band was happily detected, about the thickness of an average whip-cord, passing across the bowel, which had been drawn down and evidently strangulating it. The cause of the symptoms had thus become tolerably clear, for the strangulation of the bowel by the band explained them all, and the next question arose as to its division, for the band was placed so high up within the abdomen that it could not be brought into view, and even with scissors there would evidently be some difficulty in dividing it with safety. The abdominal opening was consequently again enlarged upwards: making the whole incision about two inches long, and the strangulated bowel brought, with its constricting band, more under control. The fibrous cord was then easily made out, for the end of the finger could be passed fairly beneath it, and Dr. Wilkinson and Mr. Giles readily satisfied themselves as to its nature. The finger of the right hand was then, as before, introduced along the bowel, which Mr. Giles kindly drew down and steadily held; its extremity was passed beneath the band, which was thus rendered tense, and a pair of scissors introduced upon the finger; the points were kept carefully closed and imbedded in its pulpy portion;—this precaution having been taken with the view of guarding the intestines which pressed around from being wounded—when the band was reached it was cautiously divided, the scissors being opened sufficiently far, and no farther than, to cut it through. The band was so gristly that its division was clearly heard, and when completely divided it gave way with a distinct noise. The strangulated bowel was thus freed at once; the wound was therefore carefully adjusted and closed with sutures, a pad of lint was applied and fixed in position by a bandage, and a suppository of opium given.

When the patient had recovered from the effects of chloroform he expressed himself as being comfortable. He had lost the pain he had endured for the three days, and was quite easy.

The following day, when I saw him with Dr. Wilkinson, at 3.30 p.m., or about twenty-four hours after the operation, I found that he had been quite comfortable since the operation,

that no vomiting or abdominal pain had taken place; he had passed a quiet night, having slept at intervals; his pulse was of good power, 90; countenance quiet and free from anxiety; tongue moist; abdomen flaccid. He had taken three grains of opium during the twenty-four hours and a little ice. Some ice and milk were ordered for him, and another pill of opium at night should it be required.

On January 3rd, or second day, he was going on as well as possible; he had passed a good night and was free from pain. He had passed flatus per anum during the night. There was no single symptom to cause alarm. The wound was healthy and had nearly healed. Beef-tea and bread were ordered.

A daily report after this date is clearly unnecessary, for everything went on as satisfactorily as could be wished. The bowels acted naturally on the evening of the third day, and again on the fourth and fifth; he then took solid food and some little wine. When I saw him again on January 8th, or the ninth day after the operation, he was convalescent; the wound had nearly completely closed, and in all other respects he was well.

*Remarks.*—I have been induced to bring this case before the notice of the Fellows of this Society from the belief that it is of sufficient rarity to be worthy of their attention, and from the feeling that it is a type of a large class of cases in which surgery has hitherto done but little; but in which I am disposed to believe that there is much to be done.

The case must be regarded essentially as one of internal strangulation of the bowel from a fibrous band; the hernia had evidently nothing whatever to do with the symptoms; it was an accidental association of the more serious condition. It is true that it is possible the presence of a hernia for twenty-five years might have led to the formation of the internal band, and have thus been an indirect cause of the strangulation; but there is no positive evidence that such was the case, for these internal bands are found at least as frequently without the existence of a hernia as with, if not more so. Practically, the two conditions must be regarded as



distinct, and as having no relation to one another. When I first saw the case, and had carefully gone into its history and reasoned upon the symptoms, I was disposed to think that it was one of two classes; that it was one of the class of cases which Mr. Birkett has so ably described in this Society, and that the strangulated bowel had been returned into the abdomen unrelieved, and was pushed upwards between the peritoneum and abdominal fascia; or that the symptoms were altogether independent of the hernia, and were due to the presence of an internal band by which the bowel was strangulated.

That the case before us belonged to the first class of cases was not very clear, for all existing evidence went to show that the hernia had nothing whatever to do with the symptoms, for there was no partial reduction and return of the hernia, no fulness in the seat of the abdominal opening, no pain even on firm pressure over the part; the hernia was also a direct one. Still, there were the possibilities of the case which raised the question. On the other hand, the evidence that the case was one of strangulation of the bowel from a fibrous band was not clear, for such cases are unfortunately always obscure; still, from the fact that strangulation of the bowel evidently existed, that the pain was fixed in one spot and paroxysmal, the suspicion of its true nature was excited; and the vivid recollection of another case of a similar kind, in which the patient died without relief, tended to confirm the idea I had originally formed.

The case to which I have alluded was the following; it occurred seven years ago.

*CASE 2.—Case of internal strangulation of the bowel by a band, associated with a scrotal hernia.*

On the 19th of January, 1860, I was called by Mr. Green, of the Old Kent Road, to see a patient, J. C—, æt. 34, who was suffering from severe symptoms of strangulation of the bowels. He had been ill sixteen hours, the attack coming on suddenly with intense pain in the abdomen, accompanied with vomiting. There was a hernia in the left side of his

scrotum, which had existed five years, and for which he had worn a truss; a small portion of the hernia, however, had always been down. When his symptoms first appeared the hernia had suddenly enlarged and he was unable to reduce it. Mr. Green was sent for, and returned a portion of the contents of the sac, but the symptoms continuing, he sought my advice.

When I saw him he was vomiting a yellow bilious fluid, and was in severe pain about the umbilicus. His abdomen was neither very tense nor very tender upon manipulation. The scrotal hernia was large, but flaccid and painless. His countenance denoted extreme anguish. Pulse small and rapid; skin bedewed with a cold sweat.

The symptoms of intestinal obstruction being thus very marked, and a hernia existing, chloroform was given, and under its influence an attempt was made to reduce the contents of the hernial sac, but with no permanent effect; for although a portion was returned and the patient asserted that the hernia had been reduced to its natural size, the symptoms persisted and soon became aggravated, paroxysms of pain attacking the patient.

Three hours subsequently herniotomy was performed; omentum was alone found in the sac, but no intestine; the mass of omentum had evidently been down for some years. The finger could be readily introduced into the abdomen, but no signs of strangulation by any of the parts which had entered into the formation of the hernia could be made out.

After the operation the patient expressed himself as being relieved. Opium was ordered in full doses.

The next morning (20th) he was more comfortable, having passed a tolerable night; he had vomited but once, an hour after the operation, and had been free from any severe paroxysm of pain; his abdomen was firm, but not much distended. He was ordered to continue the opium.

On the evening of the 21st I was hastily called to see him, as he had been again seized with a severe paroxysm of abdominal pain. I found him with a cold clammy skin, sunken eye, and almost imperceptible pulse. His abdomen

was slightly tympanitic, although not tender, for firm pressure could easily be tolerated ; he had not passed anything by the bowel, nor any urine. A catheter was passed, but only an ounce and a half was drawn off. It was too evident that he was sinking, and that any operative interference was clearly hopeless. He died eighty-eight hours after the first symptoms, and sixty-eight after the operation of herniotomy, in a severe paroxysm of pain in the epigastrium, attended with vomiting.

The necropsy revealed the true seat of the malady and that the hernia had nothing whatever to do with the symptoms. Some feet of the middle of the small intestines were found much distended, of a black colour, and almost gangrenous; they had evidently been strangulated by a small string-like band about an inch and a half long, which passed from a piece of intestine backwards to the mesentery near the promontory of the sacrum. The division of this band at once freed the bowel, and it was clear that this operation could have been readily done during life.

*Remarks.*—The analogy between the two cases I have related was so great that it was impossible for me in the first one, not to entertain the idea that the cause of the strangulation of the bowel was of the same character as the post-mortem examination of the last had revealed to me.

In that case, when it was clear that the seat of strangulation was not in the parts which formed the hernia, the idea of opening the abdomen more freely than had been done in the herniotomy, with the view of giving relief, was entertained, but unfortunately abandoned ; and when it could have been carried out the time for doing so had passed away, for the patient was at death's door. But in the successful case to which I have drawn your attention no obstacle existed to prevent the adoption of the practice which had been suggested, and the result of the treatment must be regarded as being most satisfactory.

In both cases there was distinct evidence of intestinal obstruction ; in both the pain was situated in one spot, and was of a paroxysmal nature ; in both hernia existed, but in

neither was there any pain in the tumour, nor any tenderness, even on manipulation or pressure. In the fatal case a simple enlargement of the abdominal wound upwards would have enabled me to have reached and, I take it, to have divided the band by which the bowel had been strangulated. In the successful case this practice was carried out with a good result. I am disposed to believe that in many other cases of intestinal obstruction, when the symptoms are marked, the pain fixed and paroxysmal—whether with or without a hernia—relief may often be afforded by an operation, when they are now left to die; for we know by purely medical treatment, in a large proportion of cases, little or no good is to be achieved. Even the use of opium in some has its drawbacks, for it may, as in Case 2, mask the symptoms and mislead. An exploratory incision might be made on the outer border of the rectus muscle on the side indicated by the fixed pain, and through this relief to the strangulated bowel might be given. It is foreign to my present purpose, however, to enter more fully into the subject of internal strangulation of the bowel than the cases I have brought forward appear to justify, but I may be pardoned for expressing an opinion that there is yet much more to be done by surgery in these affections than has hitherto been attempted, and a hope that the cases I have had the privilege of bringing before the Society will do something towards its realisation.



ON THE  
CONDITION OF THE URINE  
IN  
THREE CASES OF EPILEPSY.

BY  
F. W. GIBSON, M.D. LOND.,  
ASSISTANT MEDICAL OFFICER TO THE CRIMINAL LUNATIC ASYLUM,  
BROADMOOR.

COMMUNICATED BY  
WILLIAM JENNER, M.D., F.R.S.

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“THE subject of the influence of the nerves on the urinary excretion is not yet sufficiently advanced to admit of profitable discussion;”<sup>1</sup> nevertheless, the information derived from the experiments of Bernard and others would lead to the prediction of an increase in the urinary constituents, especially of the water, during the hours following an epileptic attack, for that paralysis of the sympathetic and dilatation of the small vessels of the system occur during that period is now tolerably well-established.

The following analyses in three cases of epilepsy<sup>2</sup> were undertaken with a view to determine if facts accorded with this theory. The first table contains a record of the twenty-four hours’ urinary excretion, during twenty-eight days, of

<sup>1</sup> Parkes ‘On the Urine,’ pp. 99, 100.

<sup>2</sup> In all the cases the brain alone was affected. The amount of urea and chloride of sodium was estimated by Liebig’s nitrate of mercury process; that of the phosphoric acid by the nitrate of uranium method of Neubauer.

a man, æt. 21, who suffered from epilepsy solely. He had had fits, as far as could be ascertained, during two years before his admission into the asylum in May, 1865. The number of fits recorded since that date has been on an average nineteen in the month; the majority occurred at night: not more than five have been observed during the twenty-four hours, but since he was (though very frequently visited during the night) not constantly watched, probably some escaped notice. The convulsions, though severe, did not, as a rule, continue longer than five minutes, nor did a profoundly comatose condition supervene at any time.

His daily diet during the period in which the analyses were made consisted of four ounces of cooked meat without bone, one pound of vegetables, one pound of bread, two pints of tea, half a pint of ale, and two pints of water.

The first meal was taken at eight o'clock in the morning, the principal at one o'clock in the afternoon, the last at five in the evening.

The following deductions are derived from the facts recorded in the table:

1. The mean excretion of water is slightly above the healthy average; that of the urea somewhat, that of the chloride of sodium and phosphoric acid considerably, below.

The mean<sup>1</sup> excretion per kilogramme is—water, 27·32 c.c.; urea, 0·414 gm.; chloride of sodium, 0·129 gm.; phosphoric acid, 0·029 gm.<sup>2</sup>

The man weighed 62 kilos.; was moderately muscular, and tolerably fat.

2. There is no constant change in the urine on the days on which the fits occurred.

a. The water on nine of the fit days is below the mean; on five the reduction is considerable, but can be accounted for by the great heat and dryness of those days.

On the remaining six days the water is above the average;

<sup>1</sup> Total for 62 kilos.—water, 1694 c.c.; urea, 25·6 gms.; chloride of sodium, 7·99 gms.; phosphoric acid, 1·79 gms.

<sup>2</sup> The normal mean, as given by Dr. Parkes, is—water, 23 c.c.; urea, 0·500 gm.; chlorine, 0·126 gm.; phosphoric acid, 0·048 gm.

on the day on which the largest amount was excreted two pints extra of fluid were added to the diet. Excluding these exceptional days, the mean of the water excretion on the fit days is 1935 c.c., considerably above 1075 c.c. the mean of all the low temperature days.

There would appear, therefore, to be a tendency to increase of water on the fit days.

*b.* The urea on seven of the fit days is above the mean, on seven slightly below; on none of the days save one are the maximum and minimum amounts greater or less than can be accounted for by the normal variations occurring in a healthy person, for the maximum and minimum amounts passed from day to day by such an individual are usually one fifth above or below his mean amounts.

On one day, July 20th, on which very little solid food was taken, the usual meat being replaced by two pints of beef tea, the urea during the twenty-four hours following a very severe fit amounts to 32.16 gms. On October 21st, a day not recorded in the table, on which five fits were observed, the amount of food being the same as on July 20th, the urea amounted to 28.7 gms.; but on the other hand, on July 19th, a two-fit day, the urea amounts to 24.4 gms. only, although the ordinary diet was taken. The mean of urea on all the fit days is only slightly, 0.8 gm., above the mean of all the twenty-eight days.

*c.* The normal variations in the chloride of sodium and phosphoric acid are so great that little if any value can be deduced from these columns of the table.

The mean of the former excretion on the fit days is a little greater than that of the twenty-eight days, the mean of the latter a trifle less.

Table II contains a record of the urinary excretion during thirteen days and fifteen nights. The urine of the day includes that excreted between the hours of 8 a.m. and 8 p.m. The patient went to bed at 8 p.m. and rose at 6 a.m.

The following deductions are derived from the facts stated in this table:

1. The mean of the water and chloride of sodium during



the nights is less than the mean of these excretions during the day, that of the urea nearly equal, that of the phosphoric acid greater.

2. There is no constant change in the urine of the nights on which fits are recorded, but there appears to be a tendency to increase of water, urea, and chloride of sodium.

The mean of six fit nights is—water, 825 c.c.; urea, 18.93 gms.; chloride of sodium, 4.316 gms.; phosphoric acid, 0.9842 gm.

The mean of all the nights is—water, 704 c.c.; urea, 12.21 gms.; chloride of sodium, 3.143 gms.; phosphoric acid, 1.064 gms.

The water on the fit nights is four times above, on two below the mean.

The urea is thrice above the mean.

The chloride of sodium four times above.

The phosphoric acid thrice above.

On the days preceding the fit there is no notable change in the urine, save on July 27th, on which day is a marked reduction in all the constituents. The usual diet was taken on that day.

The third table gives the mean hourly excretion for an equal number of hours before and after the fits. The facts therein recorded show that there is no constant or important change in the urine in either period.

The average of the hours before the fit is almost exactly the same as that of the hours after; hence, since the latter were fasting hours, the evidence is in favour of increase in the latter period.

The fourth table contains an account of the excretion during nine hours following a severe fit. The fit occurred at half-past eleven in the morning; no food had been taken since half-past eight.

The excretion of water, urea, chloride of sodium, and phosphoric acid, is certainly large during the hour following the fit, considering that the amount of food taken at breakfast consisted only of half a pound of bread and a pint of tea.

The amount of the constituents during the corresponding

hour after the mid-day meal, which consisted of a pint of good beef-tea with four ounces of bread, is very much less.

The urine was examined at each analysis for albumen and sugar, which were invariably absent. On two occasions only did any deposit occur; on each it consisted of urates. All, therefore, that can be fairly deduced from the results of the repeated analyses in this case is this:

“There is no constant change in any of the urinary excreta, although there appears to be some connection between the occurrence of the fits and increase in the water, urea, and (chloride of sodium?).”

Table V contains a record of the urinary excretion during fourteen nights of an epileptic man, *æt.* 33, who had had fits from a very early childhood. He is reported to have had, before his admission, as many as seven a day. Since his admission fits have been recorded at intervals of two or three days, never more than two in the twenty-four hours. The character of the attacks was very similar to that of the fits in the former case.

The urine could not, on account of his mental state, be collected during the day; he was therefore made to pass his water at eight o'clock in the evening, at which hour he went to bed, and again at eight in the morning; and the urine thus obtained was, together with that made during the night, subjected to analysis.

The last meal of the day, consisting of a pint of tea and half a pound of bread, was taken at half-past five o'clock. No food was taken during the period above named.

Deduction from this table:

There is a tendency to increase in the water on the fit nights. There is no constant change in the urea or chloride of sodium on these nights.

The mean of the fourteen nights is—water, 416 c.c.; urea, 10.75 gms.; chloride of sodium, 1.434 gm.

On all of the six nights on which fits are recorded the water is above the mean; on two considerably. On two of these nights the urea is above the mean, on one considerably. On two it is slightly below the mean, on two con-

siderably. There is no important variation in the chloride of sodium.

The mean of all the fit nights is—water, 561 c.c.; urea, 9.16 gms.; chloride of sodium, 0.915 gm.

Table VI gives the secretion of a varying number of hours before and after the fits. Table VII the mean hourly secretion on six nights of a nearly equal number of hours. Had it been possible, I should have arranged that the number should have been equal in each case.

The water in the former period is on five occasions in excess of the water in the latter; perhaps this excess is partly due to the water taken at half-past five, at the evening meal. The urea in the former period is on five occasions below that excreted in the latter; and considering that the effect of food would be more felt in the former period than in the latter, it would seem that there is a tendency to increase of urea in the hours following the fits.

I have not given the mean hourly excretion of the chloride of sodium or phosphoric acid, as no important deduction can be derived from the analyses as to the influence of the occurrence of the fits on these excretions. Neither albumen nor sugar was found at any time in this man's urine.

Table VIII shows the urinary excretion during eleven days of a male epileptic, æt. 49; he had had fits since four years of age. Since his admission in August, 1864, he has had them almost daily, sometimes as many as five of a severe character during the day, with numerous attacks of *petit mal* in addition. Occasionally, however, they intermitted for two or three days. He weighed 54 kilogrammes. His diet, save that the quantity of water was less, was the same as that of the first case.

The mean excretion per kilogramme is—water, 22.1 c.c.; urea, 0.289 gm.; chloride of sodium, 0.083 gm.; phosphoric acid (eight days), 0.027. The water is about equal to the nominal standard; the other constituents are very considerably below the mean.

Fits are recorded on three of these days; on one the water is considerably above the mean. On this day three fits

occurred; on one it is slightly below the mean, on one considerably below. On all of the days the urea is slightly above the mean.

The chloride of sodium is once considerably above the mean, once slightly above, and once slightly below. There is no important alteration in the phosphoric acid.

On the day immediately preceding that on which the three fits took place there is a marked reduction in the water, urea, and chloride of sodium.

On the day before that on which one fit took place there is a marked increase in these constituents.

I much desired to obtain analyses of the urine in those cases in which the fits take place at long intervals, and which exhibit distinct interparoxysmal periods, but I have not at present any such case amongst the epileptic population of this asylum available for the purpose of urinary analysis, since the mental state of the patients whose attacks are of this character is such as to render the collection of the urine during the paroxysmal period almost impossible.

All therefore that can be derived from the examination of these three cases is—

“There is no constant change in the urine, although there appears to be some connection between the occurrence of the fits and increase in the water and urea, and this increase is subsequent to the occurrence of the fits.”

I may state in conclusion that I have taken the temperature of a great many epileptics, both during the fits and at various other times, and that in no case of uncomplicated epilepsy have I found any deviation from the normal standard.

TABLE I.

		Urinary excretion.			
Date.	Fits, hour of occurrence.	Water.	Urea.	Chl. Sod.	Phos. Acid.
1866.		C. C.	Gms.	Gms.	Gms.
June 27—28†	12.25 a.m.†	820	24.15	8.19	1.442
28—29†	2.45 a.m.†	775	26.5	5.07	1.394
29—30	—	760	24.6	7.54	1.063
30—	—	820	28.19	6.32	2.015
July... 1	}	700	21.17	6.65	1.26
1—2		1850	29.6	12.9	1.87
2—3		1800	27.	12.6	1.35
*3—4		1000	1.	6.6	.6
*4—5†	11.45 p.m.* 1 a.m.†	900	23.4	9.	1.53
12—13†	1.30 a.m.†	800	26.4	8.8	2.08
13—14	—	800	23.2	4.8	2.
14—15	—	600	21.	6.	2.2
15—16	—	925	24.9	9.25	1.61
16—17†	1.40 a.m.†	1290	29.6	7.09	2.322
*17—18	8.50 p.m.*	1500	24.	8.25	1.71
*18—19†	11.40 p.m.* 1.20 a.m.†	3940	32.16	16.4	2.872
*20—21	10.30 a.m.*	2600	27.175	4.83	1.746
22—23	—	2950	24.	8.85	2.03
23—24	—	2940	30.71	9.787	2.323
24—25†	4 a.m.†	3250	27.725	13.29	2.23
*25—26	11.10 p.m.*	1675	23.75	7.86	1.995
26—27†	12.30 a.m.†	1665	25.475	8.237	1.652
27—28†	1 a.m.†	1780	27.7	6.83	1.691
28—29†	12.20 a.m.†	3175	29.5	9.027	1.88
29—30	—	1666	19.26	3.18	1.534
30—31	—	2555	24.437	4.487	1.836
August *6—7	1.35 a.m.*	2425	22.22	3.63	1.783
7—8	—	1470	26.28	5.88	2.068
9—10	—				

TABLE II.

DAY.					NIGHT.				
Hour of fit.	Water.	Urea.	Ch. Sod.	Ph. Acid.	Hour of fit.	Water.	Urea.	Ch. Sod.	Ph. Acid.
10.30 a.m.	C. C.	Gms.	Gms.	Gms.	—	C. C.	Gms.	Gms.	Gms.
—	2780	18.72	12.6	1.087	—	1160	13.92	5.8	1.74
—	2225	17.8	3.337	0.666	—	3375	9.375	1.5	1.08
—	2500	15.0	7.50	0.882	—	450	9.000	1.35	1.475
—	1875	15.	5.6	0.937	4 a.m.	1065	15.71	5.187	1.385
—	2025	10.575	7.665	1.05	11.10 p.m.	1225	17.15	6.125	1.225
—	1175	11.75	4.11	0.88	12.30 a.m.	500	12.00	3.75	1.1075
—	750	9.00	1.5	0.6	1 a.m.	915	16.47	6.63	1.052
—	1200	15.6	4.8	0.7	12.10 a.m.	580	12.1	2.03	0.991
—	2200	15.4	6.6	1.108	—	975	13.65	2.427	0.780
—	1200	9.6	1.8	0.5	—	460	9.66	1.38	1.034
—	—	—	—	—	11.35 p.m.	725	10.15	2.175	0.92
—	1900	13.3	2.85	0.79	—	655	11.137	1.637	1.046
—	1925	13.47	1.88	0.96	—	500	8.75	1.75	0.823
—	1700	16.38	4.68	1.108	—	300	9.9	1.2	0.96

TABLE III.

Hours.	Food.	Water.	Urea.	Chl. Sod.	Ph. Acid.	Hour of ft.	Food.	Water.	Urea.	Chl. Sod.	Ph. Acid.	Hours.
		C.C.	Gms.	Gms.	Gms.			C.C.	Gms.	Gms.	Gms.	
12	Tea 1 pint, bread 8 oz., at 8 a.m. Meat 4 oz., vegetables 16 oz., beer 1 pint, at 1 a.m. Tea 1 pint, bread 8 oz., at 5 p.m.	168	0.881	0.697	0.08	11 p.m.	Tea 1 pint, bread 8 oz., at 8 a.m.	102	1.43	0.51	0.102	12
12	Ditto	98	0.98	0.34	0.07	12 a.m.	Ditto	41	1.0	0.31	0.082	12
12	Ditto	100	1.3	0.4	0.05	12 a.m.	Ditto	48	1.0	0.15	0.082	12
6	Tea 1 pint, bread 8 oz., 5 p.m.	70	1.65	0.14	0.17	2 a.m.	None	59	0.649	0.27	0.03	6
7	Ditto	96	1.73	0.17	0.18	3 a.m.	Ditto	55	1.375	0.157	0.06	5
5	Ditto	70	1.185	0.48	0.08	1 a.m.	Ditto	80	1.5	0.6	0.08	7
3	Bread 8 oz., tea a pint, 8 a.m.	138	1.57	0.63	0.05	11 a.m.	Beef tea a pint at 1 p.m.	295	1.784	0.785	0.09	3

TABLE IV.

Hours after fit.	Food.	Water.	Urea.	Chl. of Sod.	Phos. acid.
		C.C.	Gms.	Gms.	Gms.
1	A pint of beef tea, bread 4 oz.	390	3.51	1.638	0.156
2	—	290	1.72	0.493	0.116
3	—	235	1.292	0.352	0.067
4	—	360	2.34	1.512	0.108
5	—	165	1.65	0.907	0.056
6	—	335	2.177	1.675	0.134
7	Tea a pint, bread 4 oz.	540	2.66	2.16	0.067
8	—	305	2.44	1.67	0.225
9	—	160	1.76	0.8	0.138

TABLE V.

Fits.	Water.	Urea.	Chloride of Sodium.
	Cubic cent.	Grammes.	Grammes.
1	505	9.15	0.937
2	600	17.6	0.8
2	500	10.5	1.0
2	430	11.91	1.102
—	650	15.6	2.27
—	500	10.0	1.25
—	290	9.86	1.74
—	360	10.8	1.08
1	815	7.89	1.722
2	520	7.93	1.032
—	300	11.75	3.6
—	375	7.125	0.7
—	475	9.025	0.95
—	625	9.375	1.875
Mean .....	416	10.75	1.434

TABLE VI.

Hours.	Water.	Urea.	Chl. Sodium.	Phos. Acid.	Hour of flt.	Hours.	Water.	Urea.	Chl. Sodium.	Phos. Acid.
	C.C.	Gms.	Gms.	Gms.			C.C.	Gms.	Gms.	Gms.
3	325	3.25	0.487	0.563	11.20 p.m.	9	180	5.9	0.45	0.522
7	400	4.8	0.4	0.28	3 a.m.	5	200	12.8	0.4	0.83
7	270	6.21	0.542	0.27	3	5	180	5.7	0.56	0.192
4	615	3.69	0.922	—	12	8	210	4.2	0.8	—
5	310	3.10	0.61	—	1 a.m.	7	210	4.83	0.42	—
4	475	2.85	0.385	0.145	12	8	210	5.88	0.525	0.672
6	350	7.	1.05	0.609	2 a.m.	6	210	6.33	0.42	0.437
6	510	4.08	1.02	0.21	2 a.m.	6	260	6.56	0.93	0.754
5	100	3.3	0.4	0.29	1	7	600	6.6	1.2	0.54
4	290	5.22	0.87	3.19	12	8	160	5.12	0.48	0.464



TABLE VII.

Hours.	Water.	Urea.	Hour of fit.	Water.	Urea.	Hours.
	C.C.	Gms.		C.C.	Gms.	
7	57	0.68	3 a.m.	40	2.5	5
7	37	0.88	3 a.m.	32	1.1	5
5	62	0.62	1 a.m.	30	0.69	7
6	58	1.1	2 a.m.	35	1.00	6
6	85	0.68	2 a.m.	43	1.00	6
5	20	0.6	1 a.m.	85	0.92	7

TABLE VIII.

Fits.	Water.	Urea.	Chl. of Sod.	Phosphoric acid.
	C.C.	Gms.	Gms.	Gms.
2	1075	16.5	4.75	1.307
—	1950	22.	8.5	1.57
—	1300	18.2	5.2	1.45
1	950	16.15	4.27	1.925
—	1120	20.16	5.6	1.255
—	1500	15.	3.75	—
—	660	11.22	2.31	—
—	1000	11.	2.25	—
—	900	10.8	3.6	1.087
3	1400	17.2	6.3	1.4
—	1350	13.5	4.05	1.115
Mean .....	1195	15.6	4.5	1.475
Per kilo. ....	22.1	0.289	0.083	0.027

# ACUTE POISONING BY PHOSPHORUS;

JAUNDICE;

DEATH ON THE FIFTH DAY.

FATTY DEGENERATION OF THE LIVER, KIDNEYS,  
GASTRIC FOLLICLES, PANCREAS, HEART,  
VOLUNTARY MUSCLES, &c.

BY

SAMUEL O. HABERSHON, M.D., F.R.C.P.,  
PHYSICIAN TO GUY'S HOSPITAL, ETC.

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ANNE L—, <sup>1</sup> æt. 28, a single woman, residing in Blackfriars, employed at a sewing machine, was in good health till January 10th; on the morning of that day, Thursday, feeling thirst, she rose from her bed, and drank in mistake for vinegar some rat poison which had been mixed with water and placed in a cupboard. Immediately after taking the draught severe burning pain came on in the mouth and throat; the breath was "light" (phosphorescent); violent vomiting and purging soon followed; these symptoms subsided, and the pain ceased in two hours; but she had neither dyspnoea nor cough. On Friday and Saturday, January 11th and 12th, she took several doses of salts and

<sup>1</sup> Reported by Mr. R. S. Lucas.

senna, which produced free action on the bowels, but no further evacuation took place till admission on Tuesday, the 15th. She remained free from pain, but suffered from thirst, which she relieved by drinking large quantities of water. There was a recurrence of severe vomiting, and the appetite failed. No urine had been passed for nearly forty-eight hours before admission. On Tuesday morning, 15th, about half-past eight, she was seized with violent pain in the loins, which induced her to seek relief at the hospital; she also noticed on the same morning that the stomach had begun to swell, but she had not discovered that the skin had become yellow.

*State on admission, January 15th, 1 p.m.*—She was a fairly nourished young woman, of ordinary stature, and the muscles were well developed; the mind was active, but she was restless and irritable; she complained of severe pain in the back, and of being very cold; at one time she would sit up to relieve the lumbar pain, at another would suddenly lie down, placing both hands behind the head. The skin was moist, but her face and extremities were cold to the touch; the conjunctiva and the whole body were slightly jaundiced, but on the face there were portions of the skin of a deeper and redder colour, as if from circumscribed erythema or from subcutaneous effusion. The countenance was expressive of pain; the pupils were regular and of natural size; the lips were parched; the tongue dry, but clean; no marks of corrosion were to be discovered on the tongue or fauces, and there was no sponginess of the gums. No pulse could be felt at either wrist, and the first sound of the heart was scarcely audible. The chest was resonant both before and behind, except that the dulness of the liver extended higher than normal. Slight crackling could be heard at the end of each inspiration at the base of the right lung (old pleuritic adhesion). The abdomen was somewhat distended, and it was tympanitic over its greater extent. The liver was enlarged, and could be felt distinctly nearly two inches below the ribs; it reached to the left hypochondrium. Slight pain was produced by pressure at the epigastrium.

She had been able to walk from the surgery of the hospital into the ward, a distance of about 150 yards.

After some persuasion a small pot of "Roth and Rengeisen's vermin-destroying paste" was produced, similar to the one from which the poison had been taken. The substance emitted a strong odour of phosphorus.

A hot bran fomentation was ordered to be applied to the back and to the abdomen, and a hot bottle to the feet. Brandy with milk and arrowroot were given; and as medicine, aromatic spirit of ammonia, carbonate of magnesia, and wine of opium, were administered in mucilage mixture every three hours.

At 5 p.m.—She still complained of feeling very cold, and of a constant pain in the back. No pulse could be felt at either wrist, the brachial artery beat very feebly, 72 per minute: respiration 24. The temperature after the thermometer had been a quarter of an hour in the axilla was 89·8° Fahr. She had retained the arrowroot and brandy, and felt more comfortable after them.

At 9.30 p.m.—The face was warmer, the temperature in the axilla was 91·5° Fahr., the brachial artery, 80: respiration 24. The sister of the patient stated that there had been loss of sight during the evening. There had been flatulence with "sinking sensation at the stomach," but very little pain had been experienced in the abdomen. Sudden vomiting of a dark grumous fluid came on, and the patient died almost immediately, about eight and a half hours after admission, and five days after taking the poison. During the afternoon she had passed a small quantity of urine, which contained some albumen; and under the microscope it presented a large quantity of epithelium, with casts of tubes, and a few blood-corpuscles.

*Post-mortem inspection* was made by Dr. Moxon sixteen hours after death. Patches of ecchymotic blotching were found on the abdomen and thighs; and on removing the skin for the usual inspection of the chest and abdomen, extensive ecchymosis was found in numerous patches, each about half an inch in diameter. There was no ecchymosis

in the cranium. The brain weighed 50 oz., and appeared healthy in every part.

*Chest.*—In the cellular tissue about the œsophagus there was a good deal of ecchymosis. On the right side there were slight adhesions at the lower part of the pleura, and some ecchymosis, limited to the patches of adhesions. The lungs were quite healthy, and not congested at the bases. In the bronchi near the bifurcation there was some punctiform ecchymosis; so also upon the parietal pericardium. In the right auricle there was a small blood clot, but the blood was mostly fluid; on the left side a small clot was adherent to the mitral valve. Under the endocardium there were also blotches of ecchymosis.

*Peritoneum.*—There was general injection of the serous membrane but no effused lymph, nor any injection along the lines of contact of the intestine, as in commencing peritonitis; about 15 oz. of dark yellow clear fluid were present in the serous cavity. The stomach contained a pint and a half of fluid, like soot and water, and the walls were covered with tenacious sanguinolent mucus. The duodenum was in a similar state, and contained coffee-ground substance. There was some congestion of the mucous membrane, but the redness increased in the jejunum, and became extreme, with ecchymosis in the ileum; the contents were black. In the large intestine the mucus was less tenacious, and there were a few fecal masses. There was no abrasion of the surface discovered. The liver weighed 79 oz., it was of natural shape, firm and tallowy to the touch, of a pale buff colour, with numerous small ecchymotic spots. It sank slowly in water and burnt in flame. Under the microscope the cells appeared to be wholly composed of oil-globules. The gall bladder contained 3ij of dark-coloured bile. The pancreas was redder than usual, the lobules had a natural appearance, and under the microscope the cells showed a few fat-globules. The spleen weighed 4 oz., the malpighian corpuscles were not visible, the organ was firm and rather tough.

The kidney was of a light yellow pinkish colour, the cortex

was injected, and the pyramids of a deep bright red tint. The uriniferous tubes were loaded with fat, but the stroma when washed was sound; about one tenth of the tubes escaped this fatty change. The bladder was nearly empty.

There was a small cyst in the left ovary, a false corpus luteum; the uterus was healthy; the hymen entire.

As to the microscopical appearances, the blood-corpuscles were apparently unchanged; the cells of the liver were gorged with fat, and the nuclei were lost; so also with the kidney, the uriniferous tubes were distended with highly refracting globules, which were soluble in ether; the secreting cells were lost, but the stroma of the gland was unchanged. The gastric follicles were distended, and contained numerous fat-corpuscles; the pancreatic glandular cells also presented oil-globules. In the spleen minute highly refracting granules were observed, but no cellular structure. The voluntary muscular fibre was partially degenerated: some portions presented the transverse markings, others consisted entirely of highly refracting particles; similar partial degeneration was found in the heart.

Dr. Fagge administered gr. j of the phosphorus paste to a rabbit; the animal died on the fourth day, and its structures presented a very striking contrast from those of a healthy rabbit; the liver was pale and fatty in a marked degree, and the "muscular fibres (from the thigh) had almost universally lost their striæ, and had undergone granular change." Dr. Fagge could not "satisfy himself as to there being decided change in the kidney, and the fibres of the heart, although granular, were not so decidedly different from those of the healthy heart, as were those of the voluntary muscles from the corresponding healthy tissues." Four grains of the paste destroyed a rabbit at once.

Dr. Stevenson kindly undertook the chemical analysis of some of the poison paste; the substance contained "4.18 per cent. of phosphorus, a small quantity of which was oxidized. The whole pot contained 135 grains of paste, equal to 5.64 grains of phosphorus; thus the woman took 5 grains of phosphorus at the utmost. No unoxidized

phosphorus could be detected in the stomach or its contents."

Instances of poisoning by phosphorus have been frequently recorded; but it is only of late years that much attention has been drawn to the subject. It has been regarded as an irritant poison, and in some instances it has proved fatal in a few hours. Dr. Taylor, quoting from Orfila, mentions death as having occurred in four hours; and Dr. Tüngel records an instance fatal in half an hour; in other cases the patient may survive for many months. A few years ago chronic poisoning by phosphorus, as observed in those engaged in the manufactory of lucifer matches, was described by Dr. Strohl, and afterwards by many others; necrosis of the lower jaw thus produced being attributed to changes in the periosteum:

The case before us is one of an entirely different kind; the condition being produced by a large dose of phosphorus, and the symptoms being of a general and acute character.

On the continent, poisoning by phosphorus bears almost the same proportion as to frequency with other poisons as arsenic does in England; and consequently the observations are more numerous, and the pathological appearances have been fully described. Interesting references to these observations have been made each year in the publications of the Sydenham Society; in the 'Year-Book' of 1860 a case by Lewinsky is given, in which the symptoms were vomiting and purging, jaundice, somnolence, with rapid and weak pulse; the liver was found to be large, and is spoken of as "lardaceous," and the blood as uncoagulable.

In 1862, quoting from Schmidt, the same publication states, that Lewin regards the phosphorus as being held in solution in an unoxidized state by the serum of the blood; and, that in experiments on rabbits the same kind of fatty degeneration was found as in the human subject. Wagner draws attention to the universal infiltration of almost every organ with fat, and suggests that many cases of acute steatosis of the liver may have been poisoning by phosphorus. Köhler points out also the resemblance

of acute atrophy of the liver with the degeneration consequent upon phosphorus. Again, in the 'Year-Book' of 1863, from M. Bucquoy ('Union Méd.'), a case of poisoning by phosphorus is mentioned as causing fatty degeneration of the brain itself, and it is stated that a very large quantity of phosphorus was found in the brain.

In some cases the phosphorus itself has been taken; in others the ends of phosphorus matches, and in a third class of instances phosphorus poison paste.

M. Tardieu, in an excellent work recently published, describes three varieties of acute poisoning by phosphorus; and the following is the substance of his remarks on the subject.

1. Persons poisoned by phosphorus complain of pain in the throat, and swelling of the tongue; sometimes of a painful heat, but more rarely of a burning sensation at the epigastrium. They have also a general feeling of illness, with excitement, nausea without vomiting; at length, more or less tardily, vomiting comes on; the vomited matters are mucous or bilious, rarely sanguinolent, and sometimes at the commencement slightly phosphorescent when seen in the dark.

These vomitings may entirely cease; but when they come on they generally afford a certain amount of relief; they have often followed colic and diarrhoea. The stomach and the abdomen are sensible to pressure; the features are somewhat altered; the mind is not disturbed. In the space of twenty-four to thirty-six hours the vomiting ceases; the patients are in a condition to walk or to be about; they feel some vague wandering pains in the limbs, more severe, and sometimes fixed in the kidneys. The pulse is small, depressed, and always slow. This apparent depression may be prolonged for two, three, or four days, or even longer, and at the time that the recovery seems established the poisoned persons suddenly die without having presented any marked symptom.

More frequently, however, on the second to the fourth day, jaundice appears, sometimes shown in the eyes, some-



times general; and to the symptoms already indicated are added pain in the head, sleeplessness, vesical straining with retention of urine. The urine drawn off by the catheter presents an icteric tint, and is sometimes albuminous. He says, "I have recognised this albuminuria at the same time as the jaundice, on the day following the taking of the poison. The vomitings reappear at intervals, and painful alvine evacuations sometimes of an involuntary character accompany them. Then, suddenly, an acute delirium comes on, followed by coma and by death, in the space of from six to ten or twelve days."

With very young children death happens much more rapidly, in four or six hours to one or two days, and the symptoms of poisoning are reduced to some attacks of vomiting followed by somnolence and by convulsions.

2. Other cases, which M. Tardieu regards as constituting a distinct form, he has called *nervous*, on account of the predominance of those symptoms, which are, however, far from being constant. These instances present soreness in the throat, pain at the epigastrium, and the ordinary nausea without vomiting; but at the commencement numbness in the limbs, itching, painful cramps, various disturbances of sensibility, and repeated faintings are observed. The prostration is extreme, the voice lost, the skin dry. There is no fever, but great weakness and also drowsiness. In these cases, more than in other instances of poisoning by phosphorus, there does not exist the least venereal excitement. The jaundice appears, and under the yellow skin erythematous patches show themselves. "Are these," he says, "the gangrenous tints of the skin of which some German physicians speak, and which we have not met with in any of our observations?" Towards the fifth or sixth day, sometimes later, there is violent delirium; the patients utter cries; they remain for several days in a state of violent agitation, with convulsive obstruction of the jaws and jactitation of the limbs; they then pass into coma, and death happens in about the same space of time as in the preceding variety, that is to say, from the seventh to the twelfth day;

in exceptional cases only is it deferred beyond the second week.

3. Poisoning by phosphorus has not always such a rapid course, and, in a last variety, more mild, the signs of the alteration of the blood predominate, and give it, strictly speaking, a *hæmorrhagic* character. The commencement does not differ from those which we have previously described ; but the vomitings often consist of pure blood, succeeded by loose, bloody stools with tenesmus. We find a painful swelling of the liver ; the pulsations of the heart are feeble and slow, and there is great weakness. After some days in this state an apparent improvement takes place ; from time to time colic comes on, and the stools contain a little blood. At the end of three weeks or a month hæmorrhages supervene, which recur and increase ; they come from all the passages, the stomach, the lungs, the nose, the ears, the uterus, the bladder. The blood is also rendered very fluid, and petechiæ form in the skin. At the same time a sense of suffocation, cardialgia, and a general numbness show the increasing impoverishment of the blood. The jaundice does not fail in these cases, but comes on in general slowly. The eye assumes a peculiar aspect, the blood, which forms an ecchymosis under the conjunctiva, has added to it an icteric tint. The hæmorrhages return from time to time, and we have seen them reproduced for five months. The weakness increases ; the anæmic cachexia having reached an extreme degree, produces nervous symptoms, more or less frequent, more or less severe ; and death is the termination of this slow poisoning, which in one case was prolonged for a period of eight months.

The cure of the poisoning by phosphorus, when the effects are not averted from the commencement and the symptoms have been sufficiently established, remain entirely exceptional. No antidote exists, and it is in vain that we have tried emetics, magnesia, albumen, sulphur, tonics. In those cases where death is averted the poison produces a persistent weakening or partial paralysis. M. Caussé (D. Albi) has remarked as a consecutive phenomenon

an incurable paralysis of both hands. But in forming an opinion as to the consequences and the probable termination of a case of poisoning by phosphorus, we must not lose sight of those frequent remissions, that prolonged prostration in every case, and in short the insidious slowness which may deceive even after many months those hopes which have been apparently well founded.

Such is the general purport of M. Tardieu's remarks ; in the instance recently under my care the symptoms were those of acute inflammation of the mouth and throat, then of the stomach and intestine ; but the most interesting changes were those of a physiological kind, and they arose from secondary blood changes and perverted nutrition. The patient did not succumb from the local affection of the alimentary tract, but from more hidden interference with healthy glandular growth, and from exhausted nervous power ; nearly every organ had undergone fatty change, the cells of the liver, the uriniferous tubes, the gastric follicles, the pancreatic cells, and the corpuscles of the spleen, so also the voluntary and involuntary muscular fibre ; all this indicates remarkable change in the nutritive function. Some symptoms attracted marked attention ; thus the temperature was exceedingly low when first seen nine hours before death, being only  $89^{\circ}8'$ . 2nd. There was an exhausted functional state of the vasomotor nerve ; the mind was perfectly sensible ; the patient able to walk, but still no pulse could be felt at the wrist, and the heart sounds were scarcely audible ; in this condition we have a close similarity with the collapse of acute abdominal disease. 3rd. There was comparative immunity from pain in the abdomen ; for about two hours there had been burning sensation in the mouth and throat, with only slight soreness at the scrobiculus cordis, although there was acute inflammation of the stomach and intestine ; the pain was in the loins. This tends to confirm the statement we have elsewhere made, that acute inflammation of the mucous membrane of the stomach and intestine may exist without severe suffering.

Dr. Taylor mentions an instance of a woman, aged 26,

who took a decoction of lucifer matches in coffee; neither pain nor purging was produced. In four days she appeared to have recovered, but she had an attack of epistaxis; febrile symptoms then set in, and she died in one week. In another case, a girl took some phosphorus paste; there was no sickness, and neither pain nor tenderness; the symptoms seemed slight, but she sank on the fifth day, without any marked symptoms having been produced. Death probably taking place from the enfeebled condition of the heart.

It will be well to bear in mind poisoning by phosphorus as a possible cause of some obscure symptoms of disease; as (1) acute jaundice with degeneration of the liver, acute atrophy of the gland; (2) intense exhaustion of the power of the vaso-motor nerve; (3) somnolence passing into coma, resembling uræmic poisoning; and lastly, as producing one form of acute purpura hæmorrhagica; and it is on account of its diagnostic importance, as well as from the rarity of these instances amongst us, that I have brought this single case before the Society.

It has been supposed, that phosphorus becomes oxidized before absorption, and that the phosphoric acids and hypophosphoric acids are the cause of secondary changes; these hypotheses are not confirmed by the case before us; phosphoric acid in its concentrated form induces acute inflammation of the intestinal tract, but not the peculiar conditions following the administration of the unoxidized substance. Phosphorus is soluble in oleaginous substances, and it would appear to enter the blood in an unoxidized state, possibly as has been suggested, held in solution by the serum. Again, another theory has been propounded, that phosphorus acts by the direct absorption of oxygen, and thus causes the deposition of hydrocarbons, and that the arterial blood has a venous colour. This instance did not, however, bear out that statement; Dr. Moxon informs me, that the arterial blood was normal in appearance, and the fatty changes would seem to be too extensive to be explained by direct chemical attraction of oxygen. We cannot tell the precise quantity of phosphorus taken; probably not five grains.

Dr. Taylor, in his work on poisons, recently published, quoting from Galher, mentions the smallest fatal dose known as six centigrammes : less, therefore, than a single grain : we think about three grains were taken in this case. The substance would seem to have been ordinary phosphorus, for the authority referred to states that the ends of matches made of the allotropic phosphorus have been taken without inconvenience : but the same person afterwards took "the matches of common phosphorus, and died from the effects."

As to antidotes, we do not possess them ; fatty substances and oils, from their solvent action on the poison, should not be given in the first instance ; but the poison should be removed by emetics, and alkalies prescribed, as the carbonate of magnesia and of ammonia. The exhausted state of the patient compels us to administer stimulants with bland nutrient substances. The patient whose case we have recorded was pulseless when first seen by us, and we believed that fatal syncope might take place at any moment ; she survived nine hours after admission into the hospital, and five days after taking the poison.

POISONING BY PHOSPHORUS;  
JAUNDICE;  
DEATH IN SIX DAYS.  
FATTY DEGENERATION OF LIVER AND KIDNEYS.

BY  
THOMAS HILLIER, M.D. LOND., F.R.C.P.,  
PHYSICIAN TO THE HOSPITAL FOR SICK CHILDREN, AND TO THE SKIN  
DEPARTMENT IN UNIVERSITY COLLEGE HOSPITAL.

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Received May 20th.—Read June 25th, 1867.

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THE following case, though somewhat incomplete, yet, taken in connection with those recently brought before the Society by Dr. Habershon, may be sufficiently instructive to justify my reporting it.

Cecilia F. W., æt. 4½ years, was brought to Dr. Coster, of Hanwell, on Sunday the 27th January, with the following symptoms: skin hot and dry, tongue much furred, pulse about 140, and much thirst.

Her mother gave the following history of the case: On the previous day she had left her alone in the house for a short time, whilst she went out on business. The child during her mother's absence took a box of lucifer matches from the chimneypiece, and sucked the composition from as many as

two dozen of the matches. The child's breath had an exceedingly offensive smell, and the room was scented strongly with phosphorus. The mother hoping to get rid of the poison gave the child half-a-teaspoonful of jalap, which soon acted on the bowels, causing eight or nine motions; there was no peculiar smell in them nor anything in their appearance which attracted the mother's notice. A motion which the child passed on Wednesday the 30th was streaked with blood and had an extremely offensive odour. On Monday evening, the second day after she took the poison, a tendency to vomiting set in, which was continued at intervals of half-an-hour or an hour until Tuesday morning. The vomit was yellow and was said to smell of phosphorus. Ineffectual and urgent attempts to vomit continued for two days. On the fourth day the skin and conjunctivæ were jaundiced. From the second day there was great thirst; the child was extremely restless and appeared to suffer much pain, rolling about its head and moaning. On Thursday night, 31st January, convulsions set in, and on the following day death ensued.

The treatment adopted was lime-water and milk in small quantities often repeated, and beef-tea enemata. After the third day a citrate of potash mixture was given.

*Autopsy.*—Seventy hours after death rigor-mortis still continued in the lower limbs; skin and conjunctivæ very yellow; abdomen much distended with flatus.

*Thorax.*—*Lungs* natural but paler than usual. Some hypostatic congestion of both bases.

*Heart* firmly contracted, cavities nearly empty. To the naked eye the muscular fibre appeared healthy; some yellow staining of the valves; no congelation of blood in heart.

*Abdomen.*—The small intestines contained throughout mucus mixed more or less with a dark almost black treacly-looking fluid (altered blood). The stomach contained a very small quantity of the same kind of fluid.

There was no ulceration or redness either of the stomach or intestinal mucous membrane, which was on the contrary unusually pale.

*Liver* very large but excessively pale, of a light fawn or pale chamois leather colour. The centres of the lobules were very slightly darker in tint than the peripheries. The capsule separated without tearing the liver substance. Under the microscope the hepatic cells were not easily made out, being replaced almost entirely by oil-globules of different sizes.

*Kidneys*.—On section remarkably pale with a good deal of mucus in the pelvis. The renal cells presented under the microscope an excess of fat globules.

*Spleen* looked normal.

*Brain*.—Some serous effusion into the meshes of the pia-mater, especially at the base, where the pia-mater was also a little opaque. The brain generally was less vascular than usual.

This case, though meager in its details, may prove interesting to the Fellows of the Society as an appendix to Dr. Habershon's paper.





ON THE PATHOLOGY  
AND  
TREATMENT OF CHOLERA.

BY  
GEORGE JOHNSON, M.D.,  
PHYSICIAN TO KING'S COLLEGE HOSPITAL.

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I HAVE ventured to bring this subject before the Royal Medical and Chirurgical Society in the hope and expectation that a full discussion will tend to establish something like an agreement amongst those who are competent to form an opinion, as to the pathological nature of cholera, and the principles on which its treatment should be conducted. I will proceed at once to state the main conclusions at which, after a careful study of the disease, I have myself arrived, together with the reasons which have led me to these conclusions. Upon these I invite comment and criticism.

*The phenomena of cholera result from the action of a morbid poison.*—There are very few pathologists who dispute this proposition. That the disease is portable and communicable, mainly, perhaps, by means of the discharges from the alimentary canal, is generally admitted.

The very general use of disinfectants during the recent epidemic implies a wide-spread belief that there is a material poison to be destroyed.

The doctrine of the communicability of cholera from the sick to the healthy is by no means inconsistent with the fact that cases sometimes occur where it is impossible to trace the disease to any source of infection. A peculiar but unknown state of the atmosphere may convey the disease without the agency of human intercourse.

*The poison is often swallowed, and enters the system through the alimentary canal.*—This appears to be proved by the undoubted fact that impure water is, in a very large proportion of cases, the vehicle of the poison.

*The poison is sometimes taken in with the air through the lungs.*—This is rendered nearly certain by such cases as that referred to by Dr. Wm. Budd,<sup>1</sup> in which the spread of cholera through a large workhouse was clearly traced to the gaseous exhalations from infected privies. Numerous inmates who, from various causes, had not access to the infected privies entirely escaped the disease, which created frightful havoc amongst others—the food and the water being the same for the whole establishment.

Another instance in which the poison must have been conveyed through the air is afforded by the case of the two pilots who took the disease in consequence of having their open boat towed at a considerable distance astern of the steamship *England*, on board of which cholera was raging.<sup>2</sup> The pilots were never on board the infected vessel, yet both had cholera: one man died, and both took the disease home to their families near Halifax, where no case of cholera had occurred for years before.

*In whatever way the morbid poison is received into the system, whether with the air through the lungs or with the water or food through the stomach, it enters the circulation before it gives rise to its characteristic effects.*—This propo-

<sup>1</sup> 'Association Journal,' 1854, p. 974.

<sup>2</sup> 'Statistical, Sanitary, and Medical Reports,' Army Medical Department, 1866; also 'British Medical Journal,' November, 1866, p. 505.

sition is merely the statement of a general law which is applicable to every known poison, whether mineral, vegetable, or animal, whose action is not obviously that of a merely local irritant. Even such comparatively insoluble substances as calomel and arsenic very quickly find their way into the blood and the tissues, while the more soluble and liquid poisons enter the circulation and pervade the whole system with marvellous rapidity. To deny, as some pathologists have done, that the cholera-poison enters the blood is to assert that, in the case of this particular poison, a general law of physiology is suspended.

The fact that the poison, though more commonly swallowed, is sometimes inhaled, yet that however introduced into the system it gives rise to essentially the same symptoms, would alone render it probable that the blood is the receptacle of the poison, and the vehicle by which its influence is transmitted to the various tissues and organs.

*Symptoms of invasion.*—After the introduction of the poison into the system there is a period, varying from a few hours to two or three days, during which there may be no symptoms of disease—the poison is latent; but in a large proportion of cases careful observation reveals the existence of symptoms of general constitutional disturbance, probably dependent on blood contamination. In a military report on cholera in Malta in 1865,<sup>1</sup> it is stated that out of forty-two cases which were carefully noted, these symptoms existed clearly and unmistakably in twenty-seven. The symptoms were a dull and listless appearance of the countenance and eyes; a dark rim round the orbits; a loss of all activity; want of appetite; prostration; vertigo; cramps; increased perspiration and coldness of the surface on the least exertion; thirst and general *malaise*. After these symptoms had continued for a period varying in different cases from a few hours to two or three days, diarrhoea ensued, and the disease was then said to be present. These symptoms, which mark the invasion of cholera, have been

<sup>1</sup> 'Statistical, Sanitary, and Medical Reports,' Army Medical Department, 1866; also 'British Medical Journal,' October, 1866, p. 409.

described by several of the Indian practitioners. Thus, Twining says,<sup>1</sup> "Prior to the more distinct and alarming attack, there are sometimes for a few hours, and in some cases for two or three days, symptoms of indisposition, evident not only to the patient himself but to his friends. When cholera is raging severely the disease is often ushered in by diarrhœa; at other times it begins with catarrh, nausea, and oppression at the scrobiculus cordis, which are not in an early stage to be distinguished from the slight indisposition which often precedes fever. The approach of cholera in this manner makes the patient suppose he is bilious or feverish, and, if recourse be had to some of the medicines commonly used in slight ailments of that sort, the disease is said to be caused by the dose of medicine taken, when in fact it had been insidiously making progress for some hours."

The symptoms here referred to are probably due to the influence of the morbid poison and its products in the blood upon the nervous system and other structures of the body.

Following more or less rapidly upon the symptoms of invasion, or without being preceded by such symptoms, there occur in most cases of the fully developed disease the diarrhœa stage, with vomiting and purging; the stage of collapse, and the stage of reaction, either with or without consecutive fever of variable severity and duration. To describe these stages is, on the present occasion, wholly unnecessary. I may assume that every one who hears this communication is thoroughly familiar with the phenomena of the disease. The main question which I desire to discuss with this learned Society is this, *What is the Pathology of cholera collapse?* Then, as subsidiary to this question, we have to inquire in what respect the collapse of cholera resembles, and in what particulars it differs from other well-known forms of collapse.

The chief forms of collapse—not choleraic—are these:—

<sup>1</sup> 'Clinical Illustrations of the more important Diseases of Bengal,' 2nd edition, 1835, p. 9.

1st. Collapse from loss of blood or of blood constituents, as from excessive purging.

2nd. Collapse from nervous shock, as from severe mechanical injuries, or from the pain of perforating ulcer of the stomach or bowel.

3rd. Collapse resulting from the operation of certain poisons—as tobacco, digitalis, antimony.

In what respects does the collapse of cholera differ from each of these varieties of collapse?

It differs from the results of loss of blood or of blood constituents, chiefly in these particulars.

1st. The absence of a direct and constant relationship between the loss of blood constituents and the degree of collapse. Often there is an inverse relation between collapse and loss of liquid by vomiting and purging. This is a fact admitted by every authority on the subject.

2nd. The discharges invariably continue more or less while the symptoms of collapse are passing off. Death may occur without discharges from the alimentary canal; recovery never.

3rd. In the collapse of cholera, there is blueness, more or less derangement of respiration, often urgent dyspnoea, and extreme feebleness or total absence of the pulse without syncope. In the state of collapse from profuse purging there is pallor without dyspnoea, and a tendency to faint when the head is raised.

Dr. Macpherson in his work ('Cholera in its Home') quotes from Dr. Pereira a case of poisoning by two and a half drachms of croton oil, as an example of collapse with blueness from excessive purging. In that case the symptoms of collapse began within three-quarters of an hour after the dose was swallowed, and were fully developed within an hour; yet neither vomiting nor purging occurred until half an hour later. It is probable that the blueness and other symptoms of collapse were mainly due to the fact that the oil, having entered the blood, impeded the circulation through the lungs. And this is rendered still more probable by another case recorded by Pereira, in which a

man who was poisoned by inhaling the dust of croton seeds, was "in a state of collapse," although he had no action of the bowels until several doses of castor oil had been given to him.

4th. "Again, the way in which remedies tell upon the two contrasted conditions is totally and instructively unlike. The coldness and faintness of exhaustion from loss of blood constituents are relieved at once by a glass of wine or brandy: the pulse instantly acknowledges the virtue of the stimulus. But alcoholic stimulants do not warm or invigorate, even for a moment, the patient in choleraic collapse; rather they seem to make matters worse. On the other hand, venesection has often brought marvellous relief under collapse; while to draw blood from a patient who is fainting from exhaustion would probably ensure his death, and would certainly aggravate his danger."

5th. Lastly, recovery from the collapse of cholera is often as rapid as the onset of the symptoms. Whereas, recovery from the exhausting influence of a profuse drain from the blood is always slow.

The collapse from nervous shock and that which results from the depressing influence of such poisons as tobacco, digitalis, and antimony, resemble in their main features that which follows upon an exhausting drain of blood-constituents. There is coldness and pallor of the surface, and extreme feebleness of the pulse, with a tendency to syncope. These symptoms are always more or less decidedly relieved by alcoholic stimulants, and would certainly be aggravated by loss of blood.

Now there is one condition which is common to all these forms of collapse, and that is a *defective circulation of blood*. Moving blood is the life of the body and of every organ and tissue of which it is made up. A complete arrest of the circulation involves instant death; a great impediment to the circulation brings on a state of collapse which is near to death, and which in extreme cases ends in death. This defective circulation occurs in various ways.

1st. In cases of collapse from hæmorrhage or from exces-

sive purging there is an absolute deficiency of blood-constituents in the vessels.

2. In cases of nervous shock there has been no loss of blood-constituents, but the heart's action is so enfeebled that the supply of moving blood is greatly diminished.

3. In cases of poisoning by tobacco, digitalis, or antimony, the apparent exhaustion is mainly due to the paralyzing influence of the poison upon the heart, and the consequent defective circulation.

4. Lastly, in the collapse of cholera the main and essential cause of the defective circulation is not the loss of blood-constituents, as in cases of hæmorrhage or excessive purging—not an enfeebled condition of the heart induced by the paralyzing influence of nervous shock or the action of a depressing poison, but *a mechanical arrest of the blood in its passage through the minute vessels of the lungs.*

The proofs of this proposition are so numerous, so varied, and so complete, as to render it in my judgment indisputably true. They are derived from a consideration of—

1st. The anatomical characters of the state of collapse.

2nd. The harmony of these anatomical characters with the symptoms observed during life, and with the effect of certain modes of treatment.

3rd. The analogous effect of certain accidents occurring in the human subject, and of certain experiments on animals.

I will now, as briefly as is consistent with my statements being made intelligible, refer to the chief facts and arguments upon which I rely to establish the truth of this proposition.

The chief anatomical appearances observed after death during collapse, which prove an impeded flow of blood through the lungs are these: The right side of the heart, the pulmonary artery and the large systemic veins are usually full and even distended with blood, which escapes in large amount when a vessel is punctured or when the vessels at the root of the lung are cut across. The coronary veins are much distended, and usually there are specks of



ecchymosis beneath the pericardium, the result apparently of great venous turgescence during life. The left side of the heart is comparatively empty. The minute tissue of the lung contains much less than the usual amount of blood and air. The comparative emptiness of the minute pulmonary vessels is shown by the pale look and the dry feel of the lung, but better still by its diminished weight. Dr. Parkes found the average weight of both lungs in twenty-two European soldiers who had died in collapse to be twenty-six ounces and six drachms; and he remarks "if the average weight of both lungs in adult males be considered to be forty-six ounces according to Dr. Clendinning, or forty-three ounces according to Dr. Reid, then the diminution in the weight of both lungs in cholera is shown to be either seventeen or twenty ounces."

Dr. Horace Jeaffreson, in a recently published paper,<sup>1</sup> gives the weight of the lungs in four cases of collapse. In one man the two lungs weighed nineteen and a half ounces. In three women the average weight of both lungs was twenty-three ounces.

I had few opportunities of making post-mortem examinations during the recent epidemic. In three cases of collapse the lungs were weighed: in one man they weighed seventeen ounces; in another man, seventeen and a half ounces; and in a boy aged fourteen they weighed fifteen ounces.

The diminished weight of the lungs is a pretty accurate measure of the degree of anæmia of their minute vessels. The degree of collapse of the lung, too, will be greater in proportion to the emptiness of the capillaries. After death it usually happens that more or less blood gravitates into the lower parts of the lungs, and hence it is often stated that the lungs are "congested," but a local post-mortem congestion is no index of the state of the pulmonary vessels during life.

In order to ascertain the exact condition of the lungs, heart, and great vessels during life, it is obvious that the

<sup>1</sup> 'Edinburgh Medical Journal,' December, 1866.

examination should be made as soon as possible after death. The importance of this is well illustrated by the following observation of Harvey.<sup>1</sup> He says, "I have several times opened the breast and pericardium of a man within two hours after his execution by hanging, and before the colour had totally left the face, and, in presence of many witnesses, have demonstrated the right auricle of the heart and the lungs distended with blood; the auricle in particular of the size of a large man's fist, and so full of blood that it looked as if it would burst. *This great distension, however, had disappeared next day, the blood having made its escape through various channels.*"

It is probable that, apart from the influence of gravitation, the tendency of the blood to shift its position after death will be greater in proportion to the tension of the right cavities and the pulmonary artery. So that, when the vital contractility of the small arteries has ceased, the blood is driven onwards by the resiliency of the distended pulmonary artery. The blood then flows through the pulmonary capillaries into the veins which had before been comparatively empty, and thus the equilibrium of pressure is restored by a post-mortem movement of the blood.

It is manifest that the cause and the degree of the impediment which checks the flow of blood through the lungs during the collapse of cholera can be truly estimated only by a comparison of the whole of the symptoms during life, with the whole series of post-mortem phenomena; due allowance being made for such changes in the distribution of the blood in the heart and lungs as are the result of purely physical forces acting after death.

When a body is examined several hours after death it is probable enough that much of the blood, which at the moment of death was accumulated in the right cavities and in the pulmonary artery, will have been pressed onwards through the capillaries into the veins, and even into the left side of the heart. But it is physically impossible that the

<sup>1</sup> 'Second Disquisition on the Circulation of the Blood,' Sydenham Society's Translation, p. 127.

blood in any quantity can ever take the opposite course after death, namely, from the capillaries into the pulmonary artery. Anæmia of the minute tissue of the lung after death affords positive evidence of anæmia during life, but engorgement of the lungs may be and often is the result of purely physical changes occurring after death.

When death occurs during the stage of reaction and consecutive fever, the lungs are often, though not necessarily or constantly much engorged, and their weight is increased in a corresponding degree. Dr. Jeaffreson gives the weight of the lungs in three cases fatal after reaction. In a man the two lungs weighed  $55\frac{1}{4}$  ozs. In one woman they weighed  $38\frac{1}{2}$  ozs., and in another woman 53 ozs. The capillary engorgement of the lungs sometimes comes on immediately after the commencement of reaction. There is a rise of temperature, an imperfect rally, quickly followed by hurried breathing, drowsiness, and death. The lungs are found much gorged, and these cases are sometimes reported as cases of death in collapse, with congested lungs; but that reaction had commenced is conclusively shown by the presence of bile in the small intestines.

The opposite states of the lung during collapse and reaction may be compared—the one with the pale and shrunken appearance of the hands when the vessels are contracted by cold, and the other with the florid and turgid condition of the same parts when, with returning warmth, the vessels are relaxed, and there is an unusual afflux of blood.

The anatomical condition of the lungs, the heart, and the great vessels, is in strict harmony with the symptoms observed during life. During the stage of collapse the systemic arteries are so empty of blood that the radial pulse is often not to be felt, while the venous system is so full that the whole surface of the body is more or less livid.

The lividity is usually greatest when collapse has come on suddenly with but little loss of fluid. In such cases the systemic veins are distended to their minutest extremities: on the other hand, when profuse discharges precede or

accompany collapse, the fluid contents of the vessels being much diminished, there is no fulness of the superficial veins, and therefore little or no blueness.

What then is the difference between the collapse of cholera and ordinary asphyxia? In ordinary asphyxia the systemic arteries are preternaturally distended, because imperfect aërated blood is impeded in its transit through the minute systemic vessels. Whereas, in the collapse of cholera the systemic arteries, receiving a scanty stream of duly oxygenised blood, readily transmit it, and are therefore preternaturally empty. The impeded transit of unaërated black blood through the systemic vessels, is conclusively proved by Dr. John Reid's experiments on asphyxia.

In ordinary asphyxia the patient is more or less drowsy in consequence of the circulating blood containing an excess of carbonic acid. In cholera collapse the arterial blood does *not* contain an excess of carbonic acid, and the patient is *not* sleepy; but his brain is simply inactive in consequence of the scanty supply of arterial blood which it receives.

In ordinary asphyxia the lividity of the surface is due partly to distension of the superficial veins, partly to the circulation of *black* blood through the arteries. In cholera collapse it is due to the former cause alone; when that is not present lividity is absent, and the lips of a patient in pulseless collapse are often as florid as in perfect health.

In ordinary asphyxia there is capillary engorgement of the lungs, they contain an excess of blood, and are excessively heavy. In cholera collapse the lungs are comparatively anæmic and reduced in weight. In ordinary asphyxia there is *capillary stasis* in the lungs. In cholera collapse there is an arrest of a large portion of the circulating blood *before* it reaches the pulmonary capillaries.

In ordinary asphyxia all the abdominal viscera are gorged with dark blood. In cholera collapse these organs, more especially the spleen and the kidneys, are lighter and contain less blood than usual, in proportion to the scantiness of the arterial current which has flowed through them during collapse. But there is congestion of the mucous membrane

of the stomach and intestines, owing to the afflux of blood which attends the elimination of the poison.

Before I suggest an explanation of the arrested circulation in the collapse of cholera, let me briefly point out some of the obvious and necessary results of that remarkable condition.

The stream of blood through the lungs is the channel by which oxygen is introduced into the system. And *cæteris paribus*, the absorption of oxygen into the blood, bears a direct relation to the volume of blood which circulates through the pulmonary capillaries.<sup>1</sup> Exercise quickens the circulation and the respiration at the same time. The two functions are most intimately correlated. On the other hand, if the current of blood through the lungs be reduced to one quarter or one sixth of its normal amount, as it probably is during the collapse stage of cholera, the respiration becomes feeble and shallow, and the amount of oxygen absorbed is reduced in the same ratio. As a result of this there is defective oxidation of the blood and of the tissues, diminished temperature of the body, diminished exhalation of carbonic acid, and a diminished formation of biliary and urinary constituents. So that, with an almost complete suppression of bile and urine, there is little or no evidence of their accumulation in the blood during the stage of collapse. Suppression of bile and urine is, as a rule, exactly coincident with collapse. *Before* collapse the stools are pale, in consequence of their abundant dilution with water; and the urine is scanty because its liquid parts are drained off by the bowel, just as in rheumatic fever they are drained off by the skin. The secretion of bile and urine returns with the returning freedom of the circulation. When suppression of these secretions continues after reaction, this is due to defective action of the excretory glands; and the constituents of bile and urine then accumulate in the blood.

I refer to acknowledged facts when I say that carbonic

<sup>1</sup> There was an element of truth in the doctrine of the ancient anatomists, that the arteries convey "vital air or spirits" from the lungs throughout the body.

acid, bile, and urine, are joint products of oxidation. There is a constant and most intimate bond between the lung, the liver, and the kidneys. They are physiologically correlated in a very striking manner; and their active work begins simultaneously at the moment of birth. During intra-uterine life, the lungs are entirely inactive: no air is admitted into their cells, and the blood from the pulmonary artery passes directly through the ductus arteriosus into the aorta. The kidneys and the liver are nearly as inactive as the lungs. The bladder, it is true, usually contains some urine; and the *meconium* which is contained in the intestines is a modified form of bile: but the amount of these secretions formed during intra-uterine life is infinitely small in comparison with the abundant excretion which begins immediately after birth, when, with the establishment of the function of respiration, there is an evolution of carbonic acid, and a continuous formation of the two correlative secretions—the two joint products of oxidation—bile and urine. During the collapse of cholera there is a near approach to that inactive state of the lungs, the liver and the kidneys, which is the natural condition of these organs in the foetus, and as the primary cause of their joint activity at the moment of birth, is the establishment of the process of respiration; so the essential cause of their conjointly diminished activity during cholera collapse is the partial arrest of blood in the lungs, and the consequent impairment of the function of respiration.

This explanation of the suppression of bile and urine receives confirmation from the fact, which has been noticed by many observers, that if a nursing mother falls into collapse, the secretion of milk, which is *not* a product of oxidation, continues apparently unchecked, and the breasts become painfully distended. In cases of extreme collapse the secretion of milk may be diminished or suspended in consequence of the extreme scantiness of the blood supply. In like manner, and for the same reason, the secretions from the alimentary canal may be arrested or nearly so. For the continuance of any secretion there are two essential

conditions: 1. A due supply of moving blood to the secreting tissue; 2, The presence in the blood of the constituent elements of the secretion, or the materials out of which the secretion may be formed.

Another result of defective oxidation during collapse is a diminution in the amount of fibrin in the blood and an excess of colouring matter. On the other hand excessive oxidation increases the fibrin and lessens the amount of red corpuscles.

*The diminished absorption* during collapse is obviously a result of the defective circulation. *Cæteris paribus* absorption is active in proportion to the rapidity of the blood-current. During collapse the absorption of gases by the lungs, and of liquids by other tissues and organs, is lessened in proportion to the smallness and the feebleness of the blood-stream. Without doubt, too, the absorption of materials from the intestinal canal is impeded by the outward current from the blood-vessels into the bowel. Another circumstance which influences the rate of absorption is the degree of emptiness of the blood-vessels; when, during the stage of reaction the circulation again becomes free, the comparatively empty condition of the vessels renders absorption very active, and there is a probability that morbid secretions which have been retained within the bowel may then be reabsorbed and become a source of danger.

There is no disease which serves so well as cholera to illustrate the close relationship which exists between the functions of circulation, respiration, secretion, and absorption.

Here I would suggest that during collapse the contractile power of the heart is probably impaired, not by the direct action of a poison, but in consequence of the defective supply of blood through the coronary arteries.

Reverting now to the arrest of the pulmonary circulation, can we explain it? It has been supposed by some that the blood has become so thickened by the loss of its water that it cannot pass through the minute vessels of the lungs. The objections to this theory are, that the onset of collapse is

often too sudden to be thus explained ; that the circulation is restored and the symptoms of collapse pass away, while the discharge of fluid continues ; that the symptoms of obstructed circulation bear no direct relation to the loss of fluid, often rather an inverse relation exists between them ; that the anæmic condition of the lungs in collapse proves that the obstruction does not begin in the capillaries, where it obviously must first occur if the theory in question were true. Lastly, that the blood is *not* found remarkably thickened ; on the contrary, when examined soon after death it is usually very liquid, and it escapes with great rapidity when a distended vein is punctured in opening the chest.

A comparison of the physiological symptoms of collapse with the anatomical condition of the lungs after death clearly indicates that the arrest of the circulation begins in the *minute arteries*. We know that the minute arteries have muscular walls ; that when examined under the microscope in the transparent parts of living animals (the bat's wing or the frog's foot) they may be seen, when stimulated by cold or electricity, or mechanical irritants, to contract to such a degree as to obliterate their canals, and so for a time to greatly diminish or entirely arrest the flow of blood through the parts which they supply. The experiments of Hales,<sup>1</sup> performed a century ago, prove that the arteries of animals immediately after death, so long as the vital contractility remains, have the power to resist the passage of such fluids as, by their low temperature or their irritating qualities, excite the contractility of the minute vessels.

Mr. James Blake has shown by numerous careful experiments<sup>2</sup> that certain salts injected into the veins of a dog immediately arrest the circulation through the lungs. The salts of soda have this effect. The animal dies in less than a minute, and after death the left side of the heart is found empty, while the right cavities are over-distended. When air is blown into the veins of an animal, or when, in operations about the root of the neck or axilla, it is

<sup>1</sup> 'Statistical Essays,' 1769.

<sup>2</sup> 'Edinburgh Medical and Surgical Journal,' vols. lii, liv, lvi.



accidentally sucked into the veins of the human subject, it causes great obstruction to the pulmonary circulation, and often sudden death. The left side of the heart, as in Blake's experiments, is found nearly empty, while the right cavities and the pulmonary artery are distended with frothy blood. Mr. Erichsen found, by experiments on a dog recently killed, that blood mixed with air required nearly twice the pressure to drive it through the pulmonary vessels that would suffice to drive unmixed blood through the lungs.

The results of these experiments and accidents afford illustrations of the influence which the contractility of the minute arteries exerts upon the stream of blood. The *minute arteries* alone have this contractile power, and the effect of their contraction is, not to propel the blood onwards, but to narrow the stream or to arrest it entirely, as a stop-cock lessens or stops the flow of water. Dr. Marey's most interesting book ('Physiologie Médicale de la Circulation du Sang') contains abundant proofs and illustrations of the controlling influence which the contractility of the small vessels exerts upon the blood-current. The phenomena of the pulse in health and the variations of the pulse in disease are utterly unintelligible without constant reference to the contraction of the minute arteries. The only known natural agency which has the power to stop abruptly *liquid blood* in its passage through the vessels is the contraction of the small arteries. I say *liquid blood*, for it is obvious that vessels of large size may be mechanically obstructed by emboli, and when a clot suddenly obstructs the pulmonary artery the superficial symptoms—shrinking of the features, a mixture of blueness with pallor, coldness and pulselessness—are essentially the same as those which occur in cholera collapse. Although embolism is sometimes a *consequence* of the obstructed pulmonary circulation in cholera, it certainly is not the *cause*. And I repeat that the only known agency which will explain the arrest of blood is arterial spasm, a spasm which may come and go as gradually or as suddenly as bronchial spasm, and which, like that, may be of longer or shorter duration, dependent upon the persistence of the

exciting cause. It is, of course, as impossible to demonstrate the existence of spasm of the pulmonary arteries as to demonstrate that of the bronchial tubes. In both instances the theory of spasm is an inference from a number of well-ascertained facts, and the only way in which either theory can be refuted is to show that it is inconsistent with unquestionable facts. Those who, in such a case, will be convinced by nothing short of demonstrative evidence must remain sceptical until they become endowed with supernatural powers of vision.

It is interesting to compare the phenomena of spasmodic asthma with those of cholera collapse. In both diseases spasm is the main cause of the morbid phenomena. In the one the spasm is bronchial, in the other it is arterial. In both diseases there are defective respiration and impeded circulation; in one the defective respiration is primary, and the impeded circulation is a secondary result of that; in the other the phenomena occur in the reverse order. In both diseases there is evidence of the intimate relationship which exists between the function of respiration and the circulation through the lungs. In both diseases there is feebleness of voice, and in both this is mainly due to the small volume and force of the respired current of air; "the vocal pipe, feebly blown through, refuses to speak."

The theory of arterial spasm receives striking confirmation from the effect of hot injections into the veins during collapse. It has been very generally supposed that the great relief which in most cases immediately follows this operation is due to the mere addition of liquid to the blood. All the facts and arguments which are opposed to the theory that the loss of water is the main cause of collapse render it improbable that the mere addition of water to the blood would remove that condition. Some have supposed that the salts, dissolved in the water, might exert a peculiar vitalising influence on the blood. But it has been proved that there is no deficiency of salts in the blood of cholera patients, and it has been found repeatedly that hot water alone has the same effect as the injection of a saline solution.

Dr. Mackintosh, who in the year 1832 injected 156 patients, soon ascertained that the hotter the liquid the more immediate and striking was the effect. The probable cause of the arterial spasm is the irritating quality of the cholera blood; and the probable action of the injection into the veins is twofold. The mere addition of water *at any temperature* would dilute the morbid blood and render it less irritating to the vessels. The injection of *hot water* would, in addition, have a direct effect in relaxing the arterial spasm. It has been observed that if, by any external application of heat, the patient can be thoroughly warmed, the pulse and the other symptoms of collapse are simultaneously improved.

As the effect of hot injections is to improve the pulse by relaxing arterial spasm, so the effect of alcoholic stimulants during the stage of collapse is to increase the obstruction in the lungs, and thus to lessen the volume and force of the pulse.

But, it has been asked, if the cholera poison excites spasm of the pulmonary arteries, why has it not a similar influence upon the systemic arteries? I cannot answer this question except by pointing to facts which show that the systemic and the pulmonary arteries have different vital endowments, so that blood of a certain quality may be readily transmitted by one set of vessels, yet be violently opposed by the other. It is the function of the pulmonary arteries to convey black blood, and they do it readily; but when from any cause black blood enters the *systemic* arteries, its passage through the extreme vessels is resisted, and the whole arterial system becomes in consequence distended. Again, Blake ascertained that the salts of ammonia and of potash, when injected into the veins of a dog, pass readily through the pulmonary vessels, but are resisted in their passage through the systemic vessels.

It has been said that, admitting the existence of arterial spasm, this is a result of the action of the cholera poison upon the spinal cord or upon the sympathetic ganglia. Obviously the vaso-motor nerves must have an influence upon the vascular spasm. But that the immediate cause of

the arterial spasm in the collapse of cholera is the direct action of the poisoned blood upon the inner surface of the vessels, appears to be proved by the influence of the hot injections in removing the spasm. These injections would obviously have a direct local action upon the vessels, but it is not probable that they could exert an immediate influence upon the spinal cord or the sympathetic ganglia. The contraction of the minute arteries appears to be the result of a reflex action of the nerves upon the muscular tissue, and it is analogous to the spasm of the glottis which is excited by a drop of water or a crumb of bread in the larynx. It is probable that the same morbid poison which excites the arterial spasm is the cause of the painful cramps of the voluntary muscles, and of those remarkable muscular twitchings which often occur after death.

In connection with this subject it is interesting to remark that Blake observed that when a dog has been killed by injecting a solution of a salt of either baryta, strontia, or lead, post-mortem twitchings occur, which are evidently very similar to those observed in cholera, though Blake himself makes no allusion to cholera. With reference to the question whether these muscular contractions are due to the action of the poison upon the nervous system or directly upon the muscles, Blake mentions one fact which, as he says, is in favour of the latter mode of action. "When these substances have been injected into the arteries the contractions are generally most marked in the limb through the artery of which the injection has been introduced."<sup>1</sup>

Bernard has proved that the influence of the wourara poison may be cut off from the posterior limbs of a frog by ligaturing the abdominal aorta and all the soft parts except the lumbar nerves before the poison is inserted. The effect of the poison is to destroy the power of the motor nerves to transmit either the electrical or the voluntary nervous stimulus, while the nerves of sensation and the contractile power of the muscles remain intact. A frog prepared in the manner described, and then poisoned by wourara, manifests

<sup>1</sup> 'Edinburgh Medical and Surgical Journal,' vol. lvi, p. 116.

pain when his anterior limbs are pinched, by struggling with his hind limbs, while his fore limbs, paralysed by the poison, remain quite motionless. The poison, as Bernard explains, is conveyed by the blood, and acts first upon the extremities of the motor nerves, its action spreading from the periphery towards the spinal cord but not in the opposite direction.<sup>1</sup>

*The pathological significance of the discharges from the alimentary canal.*—It seems reasonable to suppose that the choleraic discharges from the alimentary canal are analogous to the profuse sweats of rheumatic fever and to the eruption on the skin in cases of smallpox and other exanthemata. They are outward manifestations of a morbid change in the blood; and inasmuch as they are of constant occurrence during the process of recovery, we must infer that they are a necessary part of that process. It is probable that the cholera poison, having entered the blood, undergoes a rapid process of self-multiplication, and spoils certain of the blood-constituents, which are then ejected through the mucous membrane of the alimentary canal. If the action of the poison be moderate in degree the disease does not pass beyond the stage of vomiting and purging, and the severity of the attack is measured by the amount of discharge from the alimentary canal. If, however, the poison be more virulent, or the patient more susceptible of its influence, the blood speedily becomes so changed that the pulmonary vessels resist its passage, and the state of collapse occurs. The impeded circulation, in proportion to its degree, interferes with the process of elimination, and thus there may be an inverse ratio between the symptoms of collapse and the gastro-intestinal symptoms. The state of collapse, therefore, is one of great peril, and while it continues the blood is being spoiled, partly by the zymotic action of the morbid poison, partly by the suspension or rather the imperfection of the respiratory changes.

When after the elimination of the poison and its products the circulation again becomes free, the blood

<sup>1</sup> 'Leçons sur les Propriétés des Tissus Vivants,' 1866, p. 177.

contains an excess of unoxidized materials which now rapidly combine with oxygen. Bile, urine, and carbonic acid are abundantly formed, sometimes more rapidly than they can be excreted; they consequently accumulate in the blood; the lungs, the liver, and the kidneys—one or all—become gorged,\* and there is a state of *consecutive fever* scarcely less perilous than that of collapse.

I have spoken of the discharges from the alimentary canal as being a necessary part of the curative process; but are they not a source of exhaustion? Unquestionably they are, and they may be fatal by their abundance. This I suspect is not infrequent when patients living in an infected district imbibe or inhale daily fresh doses of the poison, so that the frequent discharges increase exhaustion, but leave the system still infected by renewed doses of the morbid agent.

The only way in which a diarrhoea thus excited and perpetuated can be surely and safely stopped is by the discovery and avoidance of the exciting cause in the poisoned air or water. The action of repressive drugs while the cause continues in operation is either *nil* or mischievous.

*The principle of treatment.*—Patients have recovered from cholera in all its stages under the most varied and opposite methods of treatment, and without any treatment at all. It is therefore manifest that there is a natural process of cure. In speaking of the cure it is convenient to divide the process into three stages—1st, blood zymosis; 2nd, vascular excretion of morbid products from the blood into the bowel; 3rd, gastro-intestinal evacuation by vomiting and purging.

Is there any specific cure for the disease—any means by which the blood changes can be prevented or arrested? I know of none.

Can we check the process of vascular excretion? Probably opium may have this effect, and there is reason to believe that in proportion as it does this it increases the risk of fatal collapse. Should we endeavour to increase the discharges from the blood? It is doubtful whether any

drug would have the effect, even if it were desirable, which it is not. So long as the circulation is free the amount of vascular excretion will be an exact measure of the dose or the virulence of the poison to be eliminated, just as in diabetes the measure of urine secreted is an index of the amount of sugar to be discharged. When the process of vascular excretion is checked by the arrest of the circulation, the only way in which we can render any effectual aid is by setting free the circulation. This may sometimes be accomplished by hot injections into the veins and by venesection. I have before explained the action of the venous injections. Venesection probably acts by relieving over-distension of the right cavities of the heart, and perhaps it may tend to lessen spasm. In the third stage of the natural process of cure—that of gastro-intestinal evacuation—the object is to prevent the accumulation of the morbid secretions in the alimentary canal, and to ensure their speedy expulsion. Further, to avoid narcotic and repressive means until the process of expulsion has been well-nigh completed.

I purposely avoid entering into details. If we can agree upon a principle of treatment, we may greatly assist each other in working out the details.

Many practitioners believe that by opiates and astringents in the diarrhoea stage they prevent the disease passing into the stage of collapse. Their belief would be less open to question if every diarrhoea thus treated were an early stage of cholera, which tended inevitably to pass into collapse when not arrested by remedies. But is this so? I believe not. With reference, however, to this question, rather than give my own experience, of which I have said enough elsewhere, I will refer to that report on cholera in Malta to which I have before alluded. The authors state that during the epidemic three forms of diarrhoea were met with—

1. The ordinary summer diarrhoea, which was easily checked and appeared to have no relation to cholera. The second form was characterised by painless watery purging, often associated with vomiting. It was found in every degree of intensity, and when severe was classed under choleraic diar-

rhœa. Although intractable, it evinced no tendency to pass beyond a certain point or to assume a more malignant form. The third form of diarrhœa was an intensification of the second kind, and so completely intractable that in sixty-one cases, when every possible attempt was made to check it, in none did it succeed, but it was invariably followed by full development of cholera. In the words of the authors of this most instructive report, "The second variety showed no tendency to pass beyond a certain point if not stopped. Its severe form, the third variety, was clearly an early stage of cholera; and it may be fairly questioned whether a single case was prevented developing into cholera by treatment directed towards the suppression of the intestinal flux." Upon this statement I would remark that the repressive means which were employed in these cases were probably worse than useless. If the account which I have given of the pathology of cholera be true or nearly so, then it is certain that the indiscriminate employment of opiates and astringents in the diarrhœa stage must favour the tendency to collapse by impeding the exit of the poison from the blood and from the alimentary canal. I have seen the worst symptoms of collapse follow so directly upon the arrest of a diarrhœa by opium as to render it nearly certain that the phenomena stood to each other in the relation of cause and effect.

Of cholera it may be said, as of many other acute diseases, that for the cure of most cases that are curable by any means the *vis medicatrix naturæ* will suffice. Yet there are few cases in which we cannot render some assistance, and not a few in which by a discreet co-operation with nature we may turn the scale and save a life which without our aid would have been lost.

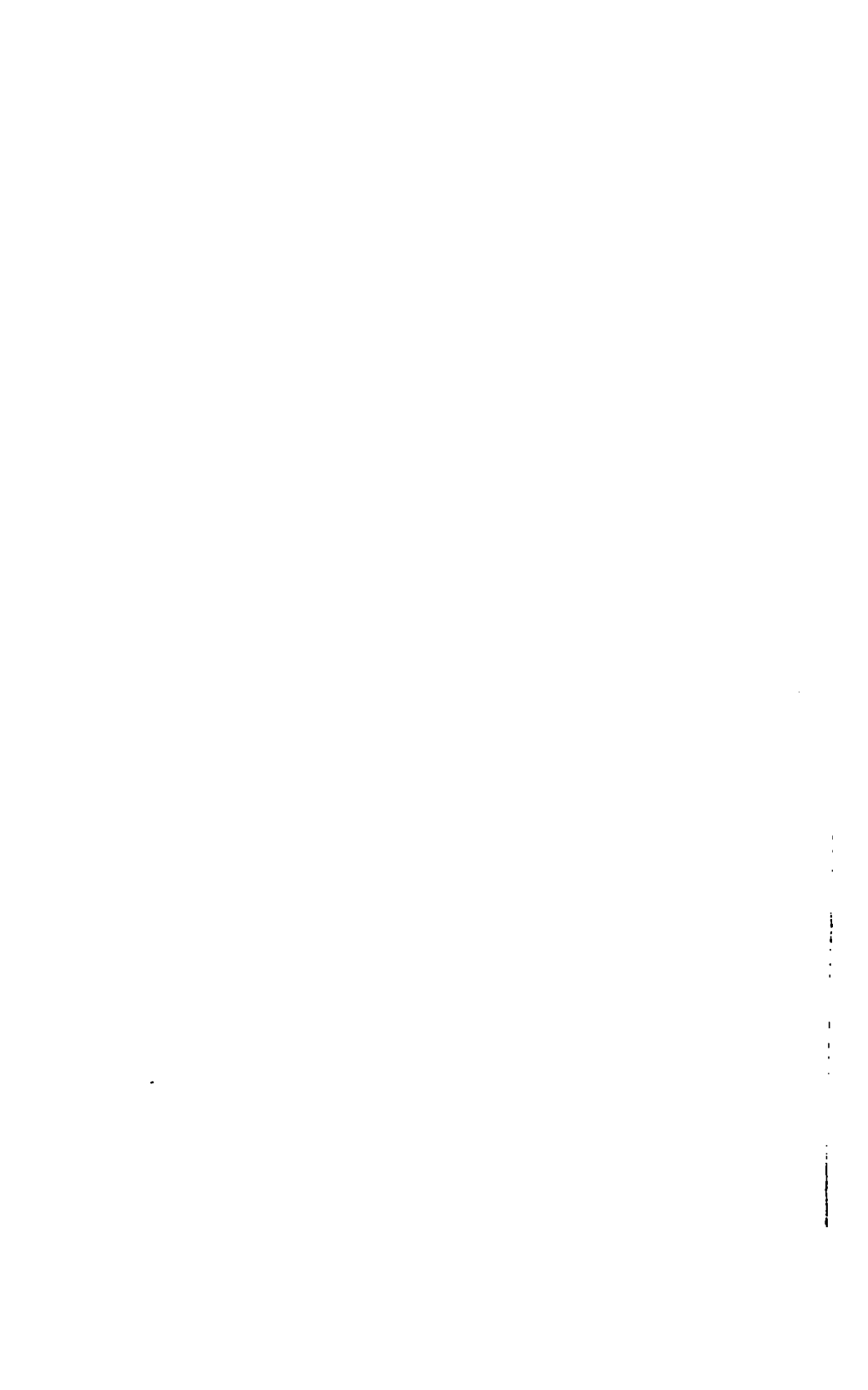
Lastly, the one great lesson which cholera is designed to teach us is a lesson of cleanliness, especially in the matter of our water supply. There is no disease which so forcibly impresses upon us the necessity of a *cathartic* method of treatment in the widest sense of that term. If we convert our rivers into sewers they must cease to be the source



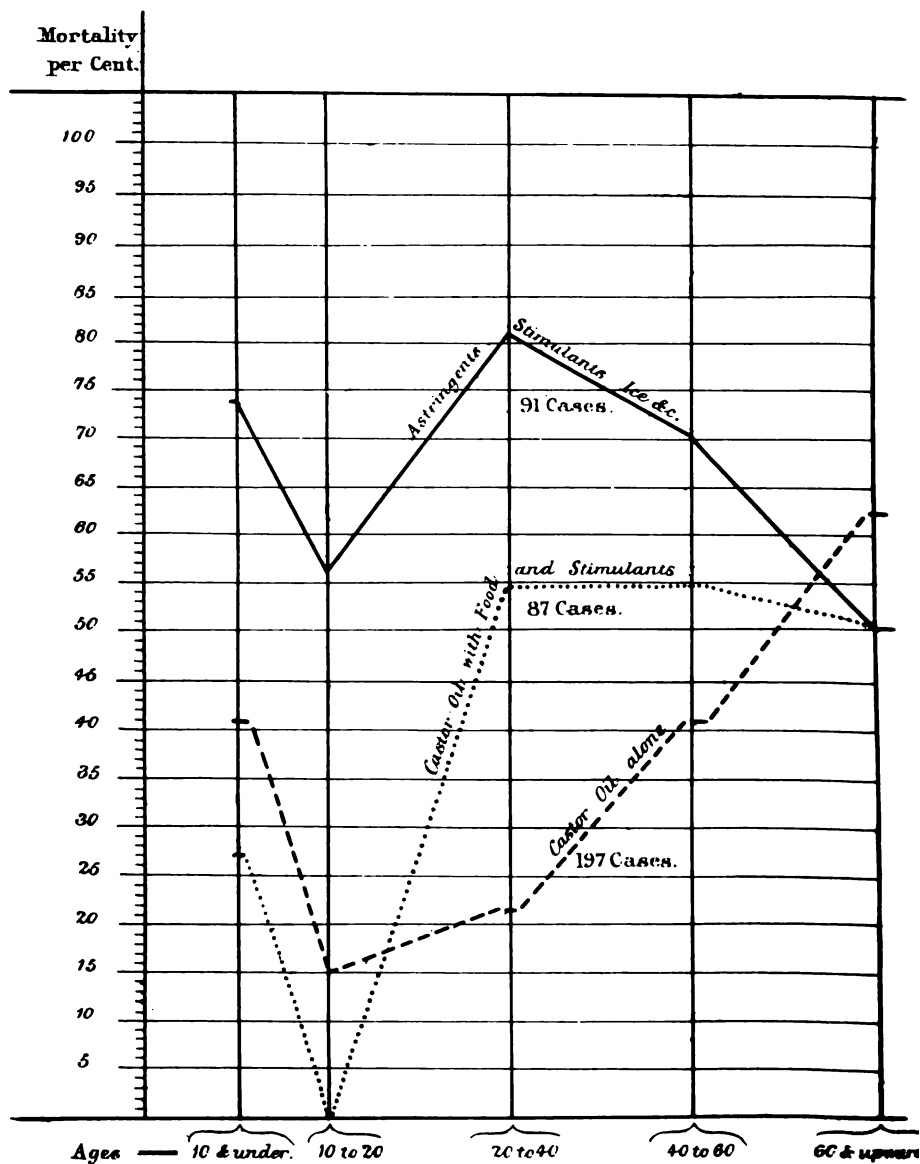
of our water supply. The philosophic Hamlet in imagination traces the dust of Alexander until he finds it stopping a bung-hole. At the present day it requires little exercise of the fancy to suppose it possible that a resident in London, on a visit to Oxford, may there deposit certain organic matters which may be restored to him in his drinking water after his return to London. It is conceivable that the same identical particle of urea might pass a second time through the kidneys of a man who drinks water from a common sewer. Unless we put a stop to this strange circulation of loathsome organic impurities our blood-vessels will refuse to transmit the intolerable stuff that we put into them. To swallow sewage, and then by repressive drugs to prevent its expulsion from the body is the very climax of irrational and destructive treatment. The drinking of sewage, however, is probably not in itself a sufficient cause of cholera; but during an epidemic season there appears to be superadded to the sewage a something which renders it a specific poison; or it may be that the effect of impure water is to render the body susceptible of the action of an atmospheric poison. It may be that the cholera poison is the product of two noxious agents, one entering the body with the air through the lungs, the other with the water through the alimentary canal.

*Postscript.*—It has been objected to the theory of arterial spasm as the cause of cholera collapse, that, if the theory were true, opium or chloroform would remove the symptoms of collapse by relaxing the spasm. This objection is based upon the assumption that these drugs have the power to relax arterial spasm; an assumption entirely unsupported by evidence. We know that there are forms of spasm over which the drugs in question appear to have no influence. Opium will not relax the spasms of tetanus; and chloroform, while it annihilates the *pains* of labour, does not arrest the *uterine contractions*. The assumption that either opium or chloroform has the power to overcome spasm of the arteries is quite gratuitous, and therefore the objection referred to has no weight.

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*Diagram (Table 4) showing the  
Mortality Per Cent, from Cholera, at different ages, and  
under different modes of treatment.*



ON THE TREATMENT  
OF  
CHOLERA AND EPIDEMIC DIARRHŒA;  
WITH A RECORD OF CASES.

BY  
J. WILSON M'CLOY, M.D.,  
LATE RESIDENT MEDICAL OFFICER, LIVERPOOL PARISH INFIRMARY, AND  
MEDICAL SUPERINTENDENT TO THE 'CHOLERA PREVENTION  
COMMITTEE,' WEST DERBY UNION;

AND  
ROBERT ROBERTSON, M.D.,  
SENIOR RESIDENT MEDICAL OFFICER, LIVERPOOL PARISH INFIRMARY.

COMMUNICATED BY  
DR. GEORGE JOHNSON.

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THERE are few subjects connected with medical science upon which greater diversity of opinion exists than upon the nature and treatment of cholera.

The disease occurs so seldom, is so obscure in the laws of its causation, so insidious in its development, so alarmingly rapid in its progress, and generally so short in its stay as an epidemic, that opportunities are wanting to investigate its nature thoroughly. Each epidemic has, however, done something towards elucidating the pathology of the disease.

From the accumulated material of the past much of the doubtful and erroneous has been removed ; facts have been retained and hypotheses inconsistent with them abandoned. Opinions that formerly it would have been heresy to question are now scouted as erroneous. Medicines that were formerly looked upon as sheet-anchors in the treatment of the disease have been laid aside with the hypotheses involving their administration. We were long taught to believe that the essential phenomena of cholera were referable to a drain of fluid from the blood produced by the intestinal evacuations. The treatment consequent upon this hypothesis was the arrest of these discharges.

Of late years, however, it has received a rude shock. It has been shown to be inconsistent with many of the acknowledged facts of the disease. Another hypothesis, which is at present engaging the attention of the medical world, attributes the collapse of cholera to a totally different cause, regards the discharges as salutary, and involves a treatment diametrically opposite to the other. Which is right ? To attempt a solution of this question we must bring both hypotheses face to face with the disease, and endeavour to ascertain which of them embraces the greater number of its facts and explains the more extensive range of its phenomena.

We had unusual opportunities during the late epidemic of observing and recording the phenomena of the disease in all its phases. Our experience we give to the profession in the hope that it may assist in placing the pathology of cholera upon a firmer basis and lead to a more rational mode of treatment. The publication of mere statistics of mortality under any mode of treatment is, for pathological purposes, useless. A record of the cases must be given showing the age, habits, &c., of the patient, his condition on admission, and the effect of the therapeutical means used ; and this as well in cases that proved fatal as in those which resulted in recovery. It would, however, be manifestly impossible in a paper like the present to give a minutely detailed record of all the cases that came under our observation, amounting, as they do, to many

hundreds. We shall therefore give in the first part of our paper a detailed record of a number of cases, embracing all the stages of the disease, and showing the general method of treatment adopted. In the concluding part we purpose giving an abstract of our cases, as tending, not only to abridge our labours, but to bring within a narrow and more distinct field of view numerous facts which in narration would extend over many pages. All the cases included in our record were treated in the Liverpool Parish Infirmary. In giving our detailed cases we selected those which would best illustrate the principles of the treatment and the main pathological facts involved in the "eliminative theory." In the abstract which follows we took the cases principally according to the dates of admission. Where there was any selection, it was only exercised in order to have as much variety, and present the disease in as many of its phases, as possible.

The number of deaths given in our record represents almost exactly the absolute death-rate under the particular mode of treatment there illustrated. The total number of admissions into the Liverpool Parish Infirmary during the epidemic was 375, and the total number of deaths 161, giving a gross mortality under all modes of treatment of 42·93 per cent. The following table shows the mortality under the different modes of treatment.

TABLE I.

Mode of Treatment.	Number of cases.	Mortality per cent.
Astringent and stimulant, camphor and ice water, applications of ice, hypodermic injections ... ..	91	71·42
Castor oil, with liberal use of food and alcohol .	87	41·37
Castor oil alone ... ..	197	30·45

The first class of cases, those which were treated by astringents and stimulants, occurred, for the most part, at the very outbreak of the epidemic, and amongst a class of

patients the most unfavorable, including drunkards, scrofulous, ill-fed youths, and foundling children (*vide* 'Lancet,' August 18, 1866, paper by Dr. M'Cloy). We cannot, therefore, attribute the high mortality under this mode of treatment to the greater severity of the epidemic at that time, because the mortality was much the same under the mode of treatment at every period of the epidemic. In the West Derby Cholera Hospital, indeed, the mortality continued almost as high as this throughout the whole epidemic. Again, the *sudden* decrease in the death-rate which followed the change of treatment pointed to the latter as being the cause of the former. The second class of cases was treated with castor oil, but large quantities of alcoholic stimulants, beef-tea, arrowroot, &c., were given during collapse. This we are now convinced was almost as faulty a combination as would have been that of castor oil with powerful astringents. The third class was treated with castor oil, receiving little or no alcoholic stimulants or food of any kind until reaction was fairly established. The second and third classes of cases were treated simultaneously.

Taking all the cases together, the mortality was greatest at the two extremes of life, as may be seen by the following table, No. II, in which is given the total number of admissions at different ages and the per-centage of deaths.

TABLE II.

TOTAL ADMISSIONS			Per-centage of deaths.		
Under	2 years		Under	2 years	
From 2—10	"	26	From 2—10	"	57·6
" 10—20	"	52	" 10—20	"	46·1
" 20—40	"	130	" 20—40	"	25·
" 40—60	"	30	" 40—60	"	36·1
Above 60	"	25	Above 60	"	52·2
Total		375	Average mortality		42·93 p. c.

Table III gives the ages of the patients treated by the three different methods, with the mortality at each period of life.

**TABLE III, showing the relative ages of the patients treated by the different methods, with the mortality at each period of life.**

Astringents, stimulants, ice, hypodermic injections, camphor, &c. &c.			Castor oil, with liberal use of stimulants, food, ice, &c. &c.			Castor oil alone.		
Age.	Cases.	Death-rate.	Age.	Cases.	Death-rate.	Age.	Cases.	Death-rate.
Under 2 years	19	73·68	Under 2 years	0	0	Under 2 years	7	14·28
From 2—10 "	10	80·00	From 2—10 "	22	27·27	From 2—10 "	20	50·00
" 10—20 "	16	56·25	" 10—20 "	10	0·00	" 10—20 "	26	15·38
" 20—40 "	21	80·95	" 20—40 "	22	54·54	" 20—40 "	87	20·68
" 40—60 "	21	71·43	" 40—60 "	31	54·84	" 40—60 "	38	39·47
Above 60 "	4	50·00	Above 60 "	2	50·00	Above 60 "	19	63·15
Total.....	91		Total.....	87		Total.....	197	
Mortality under this mode, 71·42 per cent.			Mortality under this mode, 41·37 per cent.			Mortality under this mode, 30·45 per cent.		

In Table IV (see Diagram, p. 127\*) the same results are shown by means of curved lines. It will be seen, that the line which represents the rate of mortality under the treatment by castor oil alone rises above both the other curves in the cases above the age of sixty. This is without doubt an accidental result of the small number of patients—only six—who were treated by the first and second methods. It so happened, that of the 4 cases above the age of sixty who were treated by astringents and stimulants, 2 recovered, giving a death-rate of 50 per cent., which is actually lower than that at any other period of life under the same treatment. This result, so contrary to the general experience of the relative mortality at different ages, is obviously an accident arising out of the small numbers included in that part of the series.



The two oldest patients that recovered were respectively aged seventy-one and seventy-two years. Both were admitted in collapse, both had fever during reaction, and both were treated with evacnants. The youngest child that recovered was three months old. Both its parents had previously died of cholera, and notwithstanding that it was admitted in a state of pulseless collapse, it made a rapid convalescence under aperient treatment.

In connection with the rate of mortality we may state that 11·32 per cent. of the cases admitted were moribund—dying in from ten minutes to nine hours after admission.

The rate of mortality and the mode of treatment of other cholera hospitals may be given for comparison. Almost all the cases treated in the WEST DERBY CHOLERA HOSPITAL came under our observation. These were considerably over a hundred in number. The treatment was either strongly astringent or consisted in the administration of calomel and opium. The mortality was 63 per cent. It is, however, only fair to state that many of the patients were brought very long distances in a collapsed condition, much valuable time being thus lost in the transit, and the patient rattled in a jumbling vehicle over a rough road. In the ASHFIELD STREET SHEDS the mortality was 50 per cent. The treatment adopted there was, we believe, at the first astringent. During the later months of the epidemic, castor-oil injections, administration of chloroform, hot injections into the bladder, and finally calomel in frequently repeated doses, were, we are informed, the principal means used. We have not as yet seen any details of cases or treatment from this hospital in any of the journals. We shall now give our record of cases, merely premising that the date of discharge had rarely any relation with the date of "cure." Our patients belonging to the very dregs of society, living in the lowest dens of the lowest streets, and surrounded by all the attendants of habitual drunkenness, squalid wretchedness, and abject poverty, were we considered very liable to fresh attacks of the disease or relapses, and we consequently kept

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them as long as possible in the convalescent wards, thus converted into a temporary sanatorium.

**CASE 1.—Relapse; recovery.**—M. O'N—, æt. 57, admitted September 6th, 1866, 6.45 p.m. Had been ill for two days with diarrhœa; was treated with chalk mixture, laudanum and brandy. Was seized to-day with vomiting and severe cramps.

**On admission.**—Skin cold; face livid; voice husky and almost imperceptible; pulse very weak and rapid; intense thirst; no purging or vomiting; severe cramps; considerable dyspnœa. To have  $\frac{3}{4}$ ss of castor-oil every hour. Sinapisms, dry heat, friction, &c., &c. 8 p.m.—Has been purged twice. Pulse rather better. To repeat oil.

7th.—Had five doses of oil and was purged very freely during the night; skin warmer; pulse much improved; slept a little during the night; cramps not complained of. Ordered small quantities of arrowroot, milk and wine.

8th.—Seems much better. To have another dose of oil.

9th.—Convalescent; put on full diet.

10th, 8 p.m.—Was suddenly seized a short time ago with vomiting, purging, and cramps. Is now in slight collapse. To have oil every second hour, and hot enemata every hour. Mustard sinapisms: friction with phosphuretted oil. 11 p.m.—In complete collapse: pulseless at wrists; severe cramps. Ordered five drops of chloroform every fifteen minutes for two hours. Repeated oil and injections.

11th.—Slight improvement; pulse just perceptible in radial. Vomited twice and purged freely four times during the night; passed no water; thirst intense; skin covered with a clammy perspiration. To have oil again. 8 p.m.—Improving; pulse rather fuller; has been purged three times; is drowsy. To have a little ammonia mixture.

12th, 10 a.m.—Has slept a good deal; seems much improved; no cramps or vomiting; skin warmer, but pulse still weak; passed no water. To have oil again. Ordered small quantities of arrowroot and brandy to four ounces. 10 p.m.—Improving.

13th, 10 a.m.—Is rather feverish; pulse full and soft; eyes congested; respiration considerably embarrassed; skin hot. Vomited once, purged twice; passed a quantity of high-coloured urine; great thirst still. To omit brandy and arrowroot, and have effervescing mixture every second hour. To have oil again.

14th.—Improving; purged twice during the night; evacuations horribly foetid. Not nearly so feverish. To go on with effervescing mixture.

15th.—Feverishness almost gone; passed water very freely; pulse gaining strength rapidly; was purged twice. Ordered arrowroot and milk in small quantities.

16th.—Still keeping better.

17th.—Convalescing rapidly; ordered beef tea. To have a dose of oil.

19th.—Put on full diet.

24th.—Discharged cured.

*Remarks.*—It will be remarked that the relapse in this case, if not consequent upon, at least followed the administration of nitrogenous food and alcoholic stimulants.

CASE 2.—*Collapse with subsequent relapse and recovery.*—M. C—, æt. 35, admitted September 4th, 6.30 p.m. Is of intemperate habits; had an attack of diarrhœa yesterday morning which continued all day, although she took “a good deal of cholera mixture and brandy.” Vomiting and cramps began this morning.

*On admission.*—Considerable collapse; pulse just perceptible in radial; eyes much sunken; voice husky and very feeble; temperature in axilla 95° Fahr.; extremities cold and livid; great thirst and severe cramps; no vomiting or purging. To have ℥ss castor oil every hour for three doses; to be repeated if vomited. Dry heat, sinapisms, friction, &c. 10.30 p.m.—The three doses of oil retained; no purgation; no improvement; pulse not perceptible in radial. To have another dose of oil and an enema of hot gruel, and ℥j of oil; the injection to be repeated without the oil every third hour.

5th.—Was purged very freely during the night; passed

water freely; no vomiting or cramps; pulse distinct at wrist. Inclines to sleep. Ordered a little ammonia mixture and wine.

6th.—Convalescing rapidly. To have oil.

7th and 8th.—Still doing well; pulse good; bowels have acted twice every day. Ordered beef tea and arrow-root.

9th.—Put on diet.

12th, 7 p.m.—Was seized at 5 p.m. with vomiting, purging, and cramps. Is now in slight collapse. Ordered a mustard emetic, to be followed in half an hour by ℥ss castor oil. Oil to be repeated every half hour for four doses.

13th, 10 a.m.—No improvement; has had five doses of oil; purged freely during the night; ordered a little ammonia mixture and a hot enema. 8 p.m.—Much improved; has been purged three times; pulse much stronger; general appearance better.

14th.—Improved considerably. To have a dose of oil.

15th.—Had considerable vomiting last night; is much better this morning; passed water freely twice. To have small quantities of arrowroot and brandy.

18th.—Recovering rapidly.

21st.—Put on diet.<sup>1</sup>

25th.—Discharged cured.

CASE 3.—*Great collapse; fever during reaction; recovery.*  
—C. B., æt. 35, admitted September 28th, 11 a.m. Has been ill since midnight, when she was seized with vomiting, purging, and severe cramps.

*On admission.*—In almost complete collapse. Pulse just perceptible at wrist; cramp very severe; extremities quite purple; great thirst, &c. Ordered ℥ss castor oil and ʒj turpentine, to be repeated at once if vomited, if not, in two hours. To have fifteen minims of chloroform every half hour for two hours. Dry heat, sinapisms, friction. 3 p.m.—No improvement; no purging; vomited first dose

<sup>1</sup> *Diet* includes tea, coffee, potatoes, meat, soup, &c. *Full diet* includes these with extras (*e.g.* chops, eggs, milk, &c.).

of oil. To have an emetic and two more doses of oil. To have hot injections. 11 p.m.—Vomiting frequent and cramps severe; retained one dose of oil; purged twice, discharges horribly offensive. To have oil and injection repeated. To have small quantities of beef tea.

29th, 10 a.m.—Much better; pulse distinctly perceptible. Vomiting less; skin warmer; no cramps; purged freely during night; passed no water; inclines to sleep. Ordered effervescing draught with a little brandy every second hour, and hot beef tea injections. 8 p.m.—Vomiting a little. To omit brandy and give ammonia mixture.

30th.—Rather better; skin warm and moist; inclines to doze; vomiting a little still; no purging; passed no water. To repeat oil every half hour for three doses. To have *Mist. Potas. Chlor.*

October 1st, 10 a.m.—Rather better; vomiting allayed; two doses of oil retained; purged twice during the night. To have a little brandy every second hour.

2nd.—Improving rapidly; slept well; passed water freely; purged once; no vomiting. To continue treatment.

3rd.—Considerable fever and pulmonary oppression; tongue dry and brown; passed very little water. To have one dose of oil. Ordered 4 oz. brandy.

4th.—Much the same as yesterday; still very feverish; purged once; breathing still embarrassed; passed very little water.

5th.—Feverishness and oppression still continue; eyes greatly congested; slight delirium; pulse weak and very quick; purged once; passed a little water. Ordered chlorine mixture every third hour. Blister to nape of neck and chest. Head to be shaved and cold applied.

6th.—Blisters rose well and patient seems much better; passed water freely; purged once; general appearance good.

8th.—Still improving.

9th.—Convalescent.

11th.—Put on diet.

16th.—Discharged cured.

CASE 4.—*Complete collapse, with gangrene of hands; recovery.*—J. O'B—, æt. 10, admitted August 14th, at 2.50 p.m. Was taken ill some days ago with choleraic symptoms. Treated at one of the dispensaries, and was apparently recovering. A few hours ago he had a sudden relapse: vomiting and purging set in, which was soon followed by intense algide symptoms. At this time his mother noticed his fingers and right hand rapidly alter in colour.

*On admission.*—Usual phenomena attending collapse. Fingers of right hand greatly shrivelled, and of a dark purple colour, apparently gangrenous. Discoloration, which was perfectly defined, extended on back of hand to above the wrist. On the left hand the discoloured patches were confined to the last three fingers. The mother distinctly stated that these discolorations had only made their appearance an hour or so before his admission. Ordered ℥ss oil and ℥j turpentine, to be repeated in two hours. To have a mustard emetic, hot injections, sinapisms, dry heat. Hands to be wrapped in dry cotton wool. 5 p.m.—Seems better; retained oil; bowels acted on twice. Oil just been repeated. 11 p.m.—Purged freely; vomiting and thirst less; general improvement.

15th, 10 a.m.—Considerably improved; pulse distinctly perceptible; purged twice; passed a little urine; thirst much less; right hand quite black and insensible to stimuli; fingers of left hand in the same condition. To have a dose of oil and a little beef juice. 8 p.m.—Has been purged twice; marked general improvement; sleeping.

16th.—Doing remarkably well. Right hand and wrist quite dead, also distal parts of the three fingers of left hand. Ordered wine 6 oz.

17th.—Still improving. Hands in same condition.

18th.—Going on admirably. To have one dose of oil. Ordered small quantities of beef juice and arrowroot.

20th.—Convalescent. Line of separation very distinct above right wrist and second phalanges of left hand.

24th.—Transferred to surgical wards. Amputation above

the line of gangrene separation was performed in each case by Mr. Barnes, and the boy made a rapid and complete recovery.

**CASE 5.**—*Slight collapse and recovery ; subsequent relapse and death.*—J. S—, æt. 32, admitted August 20th, at 6.45 a.m. Was seized this morning, about two hours ago, with vomiting and purging. Took nothing but a drop of brandy.

*On admission.*—Is in slight collapse ; pulse very feeble and quick ; skin cold ; vomiting a little ; complains of cramps and great thirst. Ordered ʒss of oil, to be repeated in an hour. Sinapisms, dry heat, &c. 11 a.m.—Very much better ; has retained both doses of oil and been purged four times ; vomiting ceased ; passed water freely. To have another dose of oil. 11.30 p.m.—Greatly improved ; skin quite warm ; has been purged twice since she took the oil ; passed water once.

21st.—Improving rapidly ; slept well ; purged three times during night. To have small quantities of beef tea, arrow-root, and wine. Ordered quinine mixture.

22nd.—Convalescent. Put on diet. 7.30 p.m.—After having partaken freely of “scouse”—a dish principally made from potatoes—she was seized at 5 p.m. with vomiting, purging, and cramps. Collapse rapidly setting in. Ordered ʒj of oil at once, to be repeated in two hours. To have a little ammonia mixture.

23rd.—Purged once, after both doses of oil had been given. Is now pulseless and completely collapsed. Oil to be repeated, and hot injections of oil and gruel given. Neither of these had any effect ; she rapidly sank and expired in the afternoon.

**CASE 6.**—*Great collapse ; recovery.*—M. N—, admitted August 28th, at 3.30 p.m. Husband and son died last night of cholera. Had no “premonitory diarrhœa,” but was suddenly seized about two hours ago with profuse purging, excessive vomiting, and severe cramps. She rapidly got worse,

and *on admission* was in a state of pulseless collapse; no vomiting or purging; surface of the body deadly cold and livid; had taken some medicine before she came in. Ordered half an ounce of castor oil every hour, an emetic to be previously administered. Sinapism to stomach, body to be rubbed well with oil of cajeput. 7.25 p.m.—Retained three doses of oil: has been purged twice. To have half an ounce of oil with a little ammonia mixture every two hours. Collapse still very great; pulse not perceptible in radial.

29th, 10.30 a.m.—Slight improvement; pulse feebly perceptible; skin rather warmer; breathing better; had four doses of oil, one of which was vomited; purged freely during the night; evacuated matters very offensive; has voided no urine; oil to be repeated, with ammonia mixture, every fourth hour. 3 p.m.—Better; purged twice very freely; evacuations ash green and very fœtid; no urine voided; little or no sickness. 12, midnight.—Skin warmer; pulse much stronger; purged once; vomited twice. To have a hot injection.

30th, 10 a.m.—Improving; pulse gaining strength; purged twice during night; matter yellowish green; thirst less; slept a little; breathing improved; passed a little water. Ordered an electuary containing equal parts of potass., bitart., and treacle, ʒij every three hours. To have ammonia mixture.

31st.—Improving rapidly; no thirst; not purged during the last twelve hours; tendency to sleep; passed a little water.

September 1st.—Slightly feverish this morning; breathing embarrassed; not purged since yesterday morning. To have half an ounce of oil, small quantities of brandy, arrowroot, and milk.

2nd.—Still very feverish; tongue dry and brown; vomiting twice and purged three times since oil was given; oil repeated; brandy discontinued; to have effervescing mixture *ad libitum*.

3rd.—Less feverishness; passed water freely; to continue with effervescing draught and arrowroot.



4th.—Improving rapidly ; feverishness altogether gone ; pulse gaining strength.

6th.—Had a dose of oil ; still going on favorably.

7th.—Convalescent.

9th.—Put on diet ; had a dose of oil.

14th.—Discharged cured.

*Remarks.*—The feverishness here may have been increased by the partial arrest in the eliminative process, and by the early exhibition of stimulants ; altogether, however, this was a most satisfactory case. It was undoubtedly a very severe one. About six ounces of oil were retained and very free purgation induced.

CASE 7.—*Collapse ; recovery.*—E. B—, æt. 25, admitted September 3rd, 12.40 p.m. Was ill all day yesterday with diarrhœa ; was seized this morning with vomiting and cramps, took “ mixtures ” and brandy.

*On admission.*—Considerable collapse ; skin icy cold ; *facies* and *vox cholericæ* well marked ; eyeballs sunken ; pulse small and rapid ; great thirst. To have an emetic, afterwards  $\frac{3}{4}$ ss oil every second hour. Ordered mustard poultices to calves, dry heat, friction with phosphorated oil. 5 p.m.—No improvement ; cramps very severe ; has had three doses of oil ; thirst intense ; has not been purged ; pulse almost imperceptible. Oil to be repeated with  $\frac{3}{4}$ j turpentine in each dose. 8 p.m.—Improved ; retained two doses of oil, and has been very freely purged ; skin warmer ; less lividity ; little vomiting ; passed a little water ; evacuations ash-coloured and very fœtid. To have a little ammonia mixture. 12, midnight.—Sleeping ; skin warm and moist. To have small quantities of arrowroot and beef tea.

4th.—Considerably better ; pulse gaining strength ; slept pretty well ; was purged twice freely ; passed a considerable quantity of water. Ordered wine  $\frac{1}{4}$  oz.

5th.—Still improving ; purged once.

6th.—Improving rapidly ; passed water freely. Oil to be repeated.

9th.—Convalescent ; to have beef tea and full diet.

13th.—Discharged cured.

CASE 8.—*Great collapse : recovery.*—M. M'A—, æt. 38, admitted September 7th, 4.40 p.m. Was ill for two days with diarrhœa. Was seized early this morning with vomiting and cramps.

*On admission.*—Presented all the phenomena that usually mark collapse. Pulse not perceptible in the radial. Ordered ʒss oil and ʒj turpentine every second hour. To have an injection of warm gruel. Sinapisms and dry heat to be applied. 8.30 p.m.—No improvement ; retained three doses of oil ; purged slightly once after the injection ; cramps very severe ; vomited three or four times. Oil and turpentine to be repeated. To have five drops of chloroform on sugar every fifteen minutes for three doses. Injection to be repeated. 12, midnight.—A slight improvement ; skin rather warmer ; cramps less severe ; inclines to sleep ; purged twice. To have another dose of oil.

8th.—Considerably better ; pulse weak, but distinctly perceptible ; cramps nearly gone ; purged very freely in last six hours ; vomiting ceased ; passed a small quantity of urine. Ordered small quantities of arrowroot and beef tea. Brandy to be given to 3 oz. 8 p.m.—Slept a little ; vomited after the brandy ; purged twice ; general improvement.

9th.—Going on favorably ; passed water freely ; pulse pretty good ; to have a dose of oil.

12th.—Put on diet.

15th.—Discharged cured.

CASE 9.—*Collapse ; recovery.*—J. P—, æt. 28, admitted August 11th, at 11 a.m. Has had premonitory diarrhœa for some days, for which he was treated with astringents. At 3 this morning was seized with vomiting and severe cramps ; purging ceased about mid-day.

*On admission.*—Pulseless collapse ; persistent vomiting ; no purging ; severe cramps ; intense thirst. Ordered half

ounce of oil every hour. Sinapisms, dry heat, water *ad lib.* 3 p.m.—Still vomiting freely; oil rejected as fast as given; no purging; temperature very low; extremities livid; cramps severe. To have two drops of croton oil with ten minims of chloroform on a piece of sugar, to be repeated again in two hours. 8 p.m.—Retained both doses of oil; no purging; patient expresses himself better. To have nothing but water *ad lib.*, and one hot injection. 11 p.m.—Slightly improved; pulse perceptible; has been freely purged, the matter being light-coloured and horribly foetid; still vomiting slightly.

12th, 11 a.m.—Much better; skin warm and moist; pulse greatly improved; very freely purged during the night; vomiting ceased; passed no water. To have small quantities of arrowroot and milk, and beef tea.

13th.—Greatly improved; passed water freely; bowels moved once this morning; matter semi-natural in colour and consistency. To have one dose of oil.

14th and 15th.—Going on admirably. Taking small quantities of wine.

16th.—Transferred to Convalescent Ward, and put on diet.

21st.—Discharged cured.

CASE 10.—*Collapse; recovery.*—B. D—, æt. 37, admitted August 13th, 6 p.m. Was ill with diarrhœa for three days; took no medicine. Was seized this morning with vomiting and severe cramps in stomach, legs and arms.

*On admission.*—Great collapse; almost pulseless; persistent vomiting; great pulmonary embarrassment; cramps very severe; voice inaudible; not purging. Ordered  $\frac{3}{4}$ ss of oil every hour. Sinapisms, friction, dry heat, hot enema. 11 p.m.—No improvement; vomited first two doses of oil; retained the others. Not purged since admission; cramps very severe. Ordered an ounce of oil to be given with warm gruel as an injection, and repeated in half an hour. To have another dose of oil.

14th, 11 a.m.—Rather better; pulse distinctly per-

ceptible; skin warmer; only purged once after injections; vomiting continues; passed no water. To have ten grains of calomel and a little ammonia mixture. 11.45 p.m.—No improvement; vomiting persistent; no purgation; passed no water. To have injections repeated and to take ʒij turpentine in warm milk.

15th, 11 a.m.—Much better; pulse improved; purged six times during the night; skin warmer; vomiting still continues; passed a little water. Ordered a mixture containing hydrocyanic acid, carb. soda, and ammonia. To have a little arrowroot and milk. 11 p.m.—Sickness still continues; no other change. To have ice water.

16th, 11 a.m.—Much better; slept for a few hours; pulse improving; not purged; passed a little water; vomiting unabated; thirst excessive. Ordered ʒij of turpentine in warm milk. To have injection repeated. 11 p.m.—Feverish; eyes suffused; purged freely after turpentine; vomiting ceased; to have another dose of oil.

17th.—Improving rapidly; not so feverish; purged twice; matter nearly natural. To have a little beef tea and brandy.

18th.—Improving still; passed water twice freely.

20th.—Convalescent; put on chop diet.

27th.—Discharged cured.

CASE 11.—*Extreme collapse; death.*—J. B—, æt. 36, a German, admitted September 5th, at 2 p.m. Taken ill at 6 p.m. yesterday with vomiting, purging, and cramps. Has not been purged since noon.

*On admission.*—In a state of extreme collapse. Eyes greatly sunken; face livid; tongue and breath icy cold; *Vox cholericæ* well marked; pulse totally imperceptible in radial and almost so in trachial artery; great restlessness, jactitations and pulmonary embarrassment; intense thirst; vomiting freely; no cramps just now. To have half an ounce of castor oil and half an ounce of turpentine immediately, the one to be repeated alone in an hour afterwards. Large mustard poultice to abdomen; dry heat; friction;

water *ad libitum*. 4 p.m.—Vomited both doses of oil; no purging; great restlessness; breathing very quick and gentle; pulse still imperceptible. To have  $\mathfrak{zj}$  of oil in warm gruel as an injection now, and to be repeated again in an hour. 12, midnight.—No improvement; no purging; neither of the injections returned; abdomen to be well rubbed; to have four drops of croton oil laid on tongue; vomiting abated.

6th.—Worse; was not purged during night: vomiting ceased; friction over abdomen continually; to have an ounce of oil every hour with some spirit of ammonia. Noon.—Died.

*Remarks.*—It will be observed that this patient was never purged, notwithstanding the large quantities of castor and croton oil given. No *post-mortem* was made.

CASE 12.—*Extreme collapse; death.*—M. D—, æt. 26, admitted September 18th, at 9 p.m.—Has had diarrhœa for a few days; was seized to-day at noon with vomiting and severe cramps; had been taking brandy and a mixture obtained from one of the dispensaries.

*On admission.*—In extreme collapse; was similar to Case 11, with the exception that here there were very severe cramps. Ordered  $\mathfrak{zss}$  of oil every half hour; Sinapisms to calves and abdomen; dry heat; friction; to have a little ammonia mixture. 12, midnight.—No improvement; no vomiting or purging; retained four doses of oil; cramps still severe. To have a dose of oil now and to be repeated in half an hour. Ordered five drops of chloroform every fifteen minutes for an hour; hot saline injections; to go on with ammonia.

19th.—10.30 a.m.—Still pulseless; cramps ceased; no purging; has not vomited since yesterday; passed no water since admission. 12, noon.—Rapidly sinking. 1 p.m.—Died.

CASE 13.—*Slight collapse; death.*—M. H—, æt. 48, admitted September 25, at 7 a.m.—Was seized this morning

at four o'clock with purging, cramps, and slight vomiting; had been drinking freely the day previous. No diarrhœa until this morning.

*On admission.*—In slight collapse; temperature sensibly reduced; pulse very quick and feeble; well-marked choleric voice and face; respiration rather difficult; great thirst; severe cramps. Ordered three ounces of oil every hour; sinapisms: dry heat; friction. 10.30 a.m.—Expresses himself better; cramps not so severe; but temperature has diminished and pulse become weaker, in fact, it is now just perceptible; there has been no purging or vomiting; retained three doses of oil. Ordered  $\mathfrak{zss}$  oil,  $\mathfrak{zj}$  turpentine, and  $\mathfrak{zj}$  spirit ammonia, every hour. 3.30 p.m.—Rather worse; has retained three doses of oil, &c.; has not purged or vomited; passed no water since admission; extremities and face livid. Oil to be repeated; to have hot injections every hour. 8 p.m.—No improvement; has not been purged; has retained two doses of oil; oil to be repeated; to have injections of oil and hot gruel; abdomen to be well rubbed. Midnight.—Patient completely collapsed and pulseless; no vomiting or purging; great restlessness; to have a salt and mustard emetic: to go on with injections; previous injections returned; passed no water.

26th, 10.30 a.m.—Evidently sinking fast; emetic acted very little; purged twice during night; great dyspnœa. 12 noon.—Died.

*Remarks.*—This case requires few comments. It is only necessary to observe that the patient stated he had taken "a good drop" of brandy through the night and in the morning. The quantity of oil given was very large; perhaps stronger and more irritating purgatives would have been of greater service.

*CASE 14.—Complete collapse; death.*—W. P—, æt. 40, admitted August 14th, at 9 p.m.—According to his mother's account he had been drinking hard and had had diarrhœa for nearly a week. This morning he was seized with vomiting and severe cramps. He had not been purged much since yesterday; does not well know how he felt yesterday as he was nearly drunk.

*On admission.*—In complete pulseless collapse; no vomiting or purging. Ordered ℥ss castor oil and ʒj turpentine every hour; hot saline injections; dry heat; sinapisms, &c. 12 midnight.—No improvement; vomited two doses of oil, retained one; not purged since admission. To have three doses of oil, &c., at intervals of an hour each.

15th, 11 a.m.—Seems a little better; retained the three doses of oil; bowels acted on twice; evacuations fetid and watery; pulse just perceptible; no vomiting. To have hot saline injections. Oil to be repeated with ammonia, but without turpentine. 3.15 p.m.—Not purged; had two doses of oil; severe cramps came on an hour ago. 5 p.m.—No improvement; severe cramps still; purged three times; evacuations ash-coloured, watery, and horribly fetid. Ordered injection of starch and turpentine; oil to be repeated. 11 p.m.—Purged a little after injection; pulse imperceptible; evidently sinking; great dyspnoea. To have an emetic followed in an hour by one dose of oil.

16th, 5 a.m.—Died; had not been purged since last visit.

CASE 15.—*Recovery from collapse and death during reaction.*—M. N—, æt. 20, admitted August 28th, 11.30 a.m. Yesterday morning felt a sort of faintness come over her, this was followed by vomiting and purging, which continued until about six this morning. She had been taking some medicine which stopped the purging, still she felt a deadly feeling coming on.

*On admission.*—In complete collapse; pulseless at radial; great dyspnoea; livid extremities. Ordered ℥ss of oil and ʒj of turpentine every half hour for three doses. Sinapisms, friction, dry heat, small quantities of ammonia, one hot injection. 3 p.m.—Retained the oil; purged four times, evacuations ash-coloured, watery and fetid; vomited twice; complains of cramps; temperature of body has risen a little and pulse is just perceptible at wrist. 7 p.m.—Purged six times since last visit; cramps severe. To have a little beef

tea every two hours. Small quantity of arrowroot and milk.

29th, 11 a.m.—Slightly improved ; pulse feeble ; purged freely during night ; vomited little ; passed no water since admission ; skin clammy. To have another dose of oil. 6 p.m.—Vomiting ; purged twice ; pulse rather better.

30th, 11.30 a.m.—Patient much improved this morning ; temperature considerably increased ; pulse much better ; inclines to sleep ; purged once at 5 a.m., but not since ; vomited a quantity of yellowish green matter (probably beef tea and arrowroot previously administered, J. W. M'C.) ; has passed no water yet. To have another dose of oil and turpentine, effervescing mixture every third hour. 6 p.m.—Still improving ; bowels moved twice after the oil. 11 p.m.—Doing well ; not purged since ; inclines to sleep.

31st.—Improving ; bowels moved once, stool of a brownish tint ; passed a large quantity of water ; sleeping. 8 p.m.—Feverish ; had a rigor an hour ago ; pulse full, soft and very compressible ; skin hot ; has been purged once ; vomited twice. Ordered a mixture containing liq. acet. of ammonia, chlorate of potash, and carb. soda. 11 p.m.—Much worse ; considerable delirium ; great restlessness. To have head shaved and blister to the nape of the neck. 1 a.m.—Sinking rapidly.

September 1st.—Died 8 a.m.

CASE 16.<sup>1</sup>—*Recovery from collapse ; death by coma during reaction.*—A. L—, æt. 44, admitted September 6th, 6.45 p.m. Had diarrhœa and slight vomiting all day yesterday. Seized with severe cramps this evening. Took some medicine which stopped the purging.

*On admission.*—The patient was in considerable collapse ; skin cold and clammy ; pulse very feeble and rapid ; voice almost inaudible ; eyeballs much sunken ; intense thirst ; no cramps, vomiting, or purging. Ordered ℥ss castor oil every hour for three doses. Dry heat, sinapisms, water *ad libitum*. 8 p.m.—No improvement ; no purging or vomiting ; has retained the three doses of oil ; oil to be repeated for two

<sup>1</sup> For this case we are indebted to our colleague Dr. I. de Zouche.



doses. 12 midnight.—Much in the same condition as before; no vomiting; purged three times. To have a hot injection.

7th.—Improved; skin warmer; appearance better; pulse much stronger; vomited a quantity of yellowish flocculent matter; purged twice, evacuations light coloured and offensive; passed no water. To have a dose of oil with ammonia mixture. 8 p.m.—Improving rapidly; purged once after the oil.

8th, 10.30 a.m.—Continues to improve; passed water freely; no vomiting; was not purged during the night. 11 p.m.—Not so well; eyes congested; breathing laboured; face flushed; pulse soft. Has been drowsy.

9th.—Patient very feverish; considerable dyspnœa; passed a little water; purged once. To have chlorate of potash mixture every two hours. Ordered arrowroot, beef tea, and brandy in small quantities. 8 p.m.—Fever increasing rapidly; patient very dull and stupid; respiration embarrassed.

10th.—Impossible to rouse her; is very comatose in appearance.

12th noon.—Died.

CASE 17.—*Great collapse; death during reaction.*—Wm. T—, æt. 38, admitted September 7th, 12.25 p.m. Is of intemperate habits. Was seized yesterday morning with vomiting, purging, and cramps; took some brandy. On examination this patient was found to be in an almost dying condition; pulseless at radial; extremities livid; jactitations and continual moaning; vomiting a little; cramps very severe. Ordered ℥ss oil, ℥j turpentine, and ℥j aromatic spirit of ammonia, to be repeated in two hours afterwards. Friction with oil of cajeput, sinapisms, dry heat, &c. 3.30 p.m.—No improvement; vomiting persistent; no oil retained; passed no water; pulse still imperceptible; no purging. To have an emetic, and three injections of castor oil and warm gruel at intervals of half an hour. 8 p.m.—Purged twice after injections; can just detect pulse at

radial; vomiting still persistent; cramps not so severe. 11 p.m.—To have injections of warm gruel; vomiting not so persistent. Not purged since.

8th, 11 a.m.—Slightly improved; vomiting still; no cramps; purged once during night; passed a very little water. To try oil by mouth now for three doses. 3.30 p.m.—Very restless; skin warmer; pulse distinct at wrist; troubled with hiccough; vomiting a little; did not retain any oil; has not been purged. To have injections repeated. 8 p.m.—Purged twice; great somnolency. To have a little ammonia mixture.

9th, 10.30 a.m.—Seems better; pulse improved; skin warmer and a little moist; no vomiting; purged twice; passed no water; sleeping; rather restless. To have arrowroot, beef tea and brandy in small quantities, brandy given not to exceed 4 oz. 11 p.m.—Has been purged once; still very restless.

10th, 10 a.m.—Improving in pulse; passed no water; has not been purged during night; sleeping. To have a dose of oil; linseed poultices and turpentine stupes to be applied to loins. 4.30 p.m.—Still sleeping; has passed no water; purged once; drowsy and stupid-looking; breathing difficult.

11th, 10.30 a.m.—Patient in a semi-comatose condition; stertorous breathing; face flushed; eyes congested; rather delirious; no vomiting or purging; has not passed any water. Ordered blister to nape of neck. To have  $\frac{3j}{\text{ss}}$  brandy every three hours. 9 p.m.—Died; had a little rigor about an hour previously.

*Remarks.*—There are two points worthy of notice in this case. The length of time the patient was in collapse, and the total amount of urine voided. We believe, as we shall hereafter point out, that the longer a patient remains in collapse the more liable is he to pulmonary congestion during the period of reaction. The administration of stimulants, we are now convinced, not only increases *this* risk but *also* that of uræmic poisoning.

CASE 18.—*Collapse : death from pneumonia during convalescence.*—E. E—, æt. 16, admitted October 1st, at 7.30 p.m.—Was seized this morning with vomiting and purging, which was soon followed by severe cramps; she believed that she was well yesterday; cholera prevailed in the street where she lived; does not know whether she had been in any of the houses where there was cholera; took four doses of a medicine which she got at the dispensary. At the time of her admission she was in a state of collapse; pulse very feeble; voice husky and weak; skin very cold; intense thirst; sinking of eyeballs; persistent vomiting; no purging. Ordered  $\frac{3}{4}$ ss of oil to be repeated again in two hours if retained, but at once if first dose be vomited; to have a little ammonia mixture every second hour. 11 p.m.—Retained both doses of oil; not purged; to have an injection.

2nd, 10 a.m.—No improvement; no vomiting; purged twice during night; to have oil repeated and hot beef-tea injections every two hours. 8 p.m.—Slightly improved; retained a dose of oil; to continue injections.

3rd. Improved; skin warm; pulse stronger; purged once; no vomiting. 8 p.m.—Much the same; to have four ounces of wine.

4th. Rather feverish this morning and very restless; pulse full and soft; has not been purged; no vomiting; passed no water since admission; to have another dose of oil. Ordered a blister to nape of neck; small quantities of arrowroot and milk with wine. 11.20 p.m.—Still very restless; has passed no water; not purged.

5th. Very feverish; tongue dry and brown; great restlessness and frequent jactitations; passed no water; to have another blister to neck. 10.40 p.m.—No improvement; dyspnœa; restlessness; has passed no water.

6th. Sinking; very feverish; to have turpentine stupes and poultices to loins. Ordered *Mist. Pot. acet.* 11 p.m.—Has not passed any water; has not been purged; still very restless. To have a dose of oil.

7th. No improvement; has passed no water; takes no

notice of what is passing; was purged once during night; eyes greatly congested; has passed no water. Ordered a leach to each temple. 8 p.m.—Could not get any leeches to bite; no improvement.

8th. Improved a little; pulse a little stronger; passed a little water; purged once. To have injections of beef tea and Condry's fluid.

9th. Rather better; passed a little water; pulse stronger.

10th. Better; pulse much stronger; passed water.

11th. Improving rapidly; purged twice, motions natural; passes water freely and regularly.

12th. Still doing well; to have quantity of beef-tea and arrowroot increased.

13th, 11 a.m.—Convalescent; sitting up in bed. 8 p.m.—Having got out of bed and walked about the room during the nurse's absence she was suddenly seized with a pain in the chest and a feeling of chill; she was put to bed, but, immediately after, her breathing became hurried and difficult, her pulse rapid and her face flushed. Crepitation heard over the bases of both lungs; to have a mustard jacket. Ordered ammonia mixture and brandy every two hours.

14th. Much worse; no respiratory sounds at base of right lung; crepitation heard high up in both lungs.

15th. Getting worse; lungs becoming more and more obstructed; counter irritation to chest. Brandy increased.

16th. Much worse; respiration almost stopped; crepitation at apices of both lungs and slight râles at bases. 11.30 p.m.—Died.

*Autopsy twelve hours after death.*—Inferior half of both lungs infiltrated with purulent matter, above this hepatization and intense congestion; several pleuritic adhesions and a small quantity of sero-purulent fluid in the cavity of the pleura; brain greatly congested; pale patches here and there on the intestinal mucous membrane, which was otherwise normal; kidneys apparently healthy; bladder con-

tained a considerable quantity of urine which was very ammoniacal.

*Remarks.*—This case shows very clearly how essential it is for the patient to be kept quiet during convalescence. The slightest excitement or irregularity of any kind, at this time, may be productive of the most serious results.

CASE 19.—*Slight attack of cholera; recovery; relapse; death probably from uræmia.*—A. J. E—, æt. 12, admitted September 4th, at 7.30 p.m.—Parents died of cholera a few days ago; was taken ill with vomiting and purging a few hours ago; took some “medicine.”

*On admission.*—Case considered “choleraic diarrhœa; there was no collapse. Ordered a mustard emetic to be followed by four two-drachm doses of oil at intervals of half an hour each; dry heat; sinapisms.

5th. Has been purged very freely and is much better; to have a little arrowroot and milk with a little port wine added to it.

6th. Improving.

7th. Sent to convalescent ward and put on diet.

11th, 11 a.m.—Early this morning was seized with severe cramps and purging; at present is bordering on collapse. Ordered  $\mathfrak{z}$ ss oil and  $\mathfrak{z}$ j turpentine to be given at once; if not retained an injection of oil and warm gruel to be given; to have a small quantity of ammonia mixture. 3 p.m.—In complete pulseless collapse; oil was not retained; was purged once after injection; to repeat injections every hour; oil to be again tried by mouth. 8 p.m.—Much in same condition; retained oil and injections but has not been purged.

12th. Slightly improved; pulse just perceptible; intense thirst and persistent vomiting; purged twice. Ordered 15 minims of chloroform every fifteen minutes for two hours; to have a hot injection. 3 p.m.—Improving; to have a little ammonia mixture every two hours.

13th. Still improving; vomiting and thirst abating; inclines to sleep; pulse still feeble. Ordered wine, arrow-

root and milk, in small quantities; passed water this morning.

14th. Still doing well; has not been purged since 12th; to have oil.

15th. Improving; passed water freely; has not been purged; vomiting ceased; to have oil again. Ordered chlorate of potash mixture.

16th. Sleeping constantly; rather feverish; passed little water.

18th. Feverishness continues; eyes congested; tongue dry and brown: very drowsy; passed no water.

19th. Very feverish; not purged; passed no water; to have oil. Ordered febrile mixture every hour.

20th. Has not been purged; passed no water. 12 noon.—Had a strong convulsion and died immediately afterwards.

*Remarks.*—There is little doubt that this patient died from uræmic poisoning. We cannot tell how much arrow-root, beef tea, &c., were given as the instructions were "small quantities and often."

TABLE II.—*Abstract of 100 cases of Cholera and Epidemic Diarrhœa, treated in the Liverpool Parish Infirmary, during the months of July, August, September, October, and November, 1866.*

No.	History, &c. &c.	Symptoms on admission.	Treatment.	Result.	Remarks.
1	J. B—, æt. 14, admitted 26th July. Inmate of Epileptic Ward of the Infirmary. Was eating in refuse out of one of the refuse tubs, shortly after which he was seized with choleraic symptoms.	Vomiting freely; cramps in abdomen and legs; twitchings in arms; pulse very weak; skin cold, and eyeballs sunken; purged while being undressed.	Free action of bowels kept up by small and frequently repeated doses of castor oil.	Cured.	Convalescent on the fourth day.
2	T. W—, æt. 13, admitted 26th July. Ill with diarrhœa for twelve hours, which came on very suddenly. Took some medicine, but does not know what it was.	"Choleroïd diarrhœa."	Had ʒij castor oil in small doses, and was very freely purged in first two days.	Cured.	Discharged on the third day.
3	M. G—, æt. 24, admitted 30th July. Has been ill since yesterday morning, when she was suddenly seized with choleraic symptoms. Had been eating potatoes for supper.	Slight collapse; no vomiting or purging; complains of cramps and great thirst.	Free purgation induced by castor oil, and kept up until the third day.	Cured.	Had altogether about nine ounces of oil, and was convalescent on the third day.

4	M. H—, æt. 12. Admitted 30th July. No history.	Great collapse; respiration very difficult; jactitations and great restlessness; no vomiting or purging; seems comatose.	Castor-oil in $\frac{3}{4}$ ss doses every quarter of hour for four doses. Injections of oil. Friction, &c.	Died.	Died a few hours after admission; was very freely purged about half an hour before death.
5	W. P—, æt. 26, admitted 30th July. Was seized this morning with choleraic symptoms. Was drinking the day previous.	Extreme collapse; no vomiting or purging; pulse imperceptible; algidæ symptoms very intense.	Had in first twenty-four hours six ounces of oil, and was purged twelve times.	Died.	On the evening of the second day he was sitting up in bed drinking beef-tea, when he suddenly fell back and died shortly after. This patient had a severe attack of rheumatic fever about twelve months previous.
6	M. M—, æt. 40, admitted 31st July. A convalescent from fever. Was seized this morning with vomiting, purging, and cramps. Had been eating pea-soup.	Slight collapse; vomiting a little; no purging; pulse very weak; great sinking of eyes; is emaciated.	Free action of bowels kept up by castor oil for two days.	Cured.	Had two and a half ounces of oil. Was convalescent on the third day.
7	T. H—, æt. 40, admitted 31st July. Was taken ill with choleraic symptoms at 3 o'clock this morning. Took a quantity of hot gin.	Was admitted at 6 a.m. in a state of collapse; pulse just perceptible in radial; extremities very cold and livid; complains greatly of cramps.	Had $\frac{3j$ castor oil on admission. 11 a.m., purged once. To repeat oil every hour. A free action of the bowels was kept up this and the following day. Third day ordered effervescent mixture as he was slightly feverish. To have <i>Pil. Rhei Co. grs. v. omnes nocte.</i>	Cured.	On the third day reactionary fever began to manifest itself. It continued for nearly ten days, during which the patient was taking febrile mixtures, opiate, &c. Counter-irritants were applied to head. When the fever began to abate recovery was very rapid.



PART II.—*Abstract of 100 cases of Cholera and Epidemic Diarrhœa, treated in the Liverpool Parish Infirmary, during the months of July, August, September, October, and November, 1866.*

No.	History, &c. &c.	Symptoms on admission.	Treatment.	Result.	Remarks.
1	J. B—, æt. 14, admitted 26th July. Inmate of Epileptic Ward of the Infirmary. Was eating in refuse out of one of the refuse tubs, shortly after which he was seized with choleraic symptoms.	Vomiting freely; cramps in abdomen and legs; twitchings in arms; pulse very weak; feet cold, and eyeballs sunken; purged while being undressed.	Free action of bowels kept up by small and frequently repeated doses of castor oil.	Cured.	Convalescent on the fourth day.
2	T. W—, æt. 13, admitted 26th July. Ill with diarrhœa for twelve hours, which came on very suddenly. Took some medicine, but does not know what it was.	"Choleroïd diarrhœa."	Had $\frac{3}{ij}$ castor oil in small doses, and was very freely purged in first two days.	Cured.	Discharged on the third day.
3	M. G—, æt. 24, admitted 30th July. Has been ill since yesterday morning, when she was suddenly seized with choleraic symptoms. Had been eating potatoes for supper.	Slight collapse; no vomiting or purging; complains of cramps and great thirst.	Free purgation induced by castor oil, and kept up until the third day.	Cured.	Had altogether about nine ounces of oil, and was convalescent on the third day.

12	E. T—, æt. 30, admitted 5th August. No history.	In considerable collapse; slight purging and vomiting; is very restless.	Ordered three 3ss doses of oil in first two hours. A third repeated until two doses are retained. This was at the end of the eighth hour, when free purging was produced. An injection of oil and warm gruel was then given, after which very free purgation took place, and the patient began to recover.	Cured.	A very moderate quantity of oil in this case produced the desired effect. Discharged on third day.
13	T. M—, æt. 26, admitted August 6th. Is of intemperate habits. Was seized with vomiting, purging, and cramps two days ago. Took some "cholera mixture" and brandy.	Vomiting freely; no purging; pulse almost imperceptible; skin very cold; extremities livid; severe cramps.	Had an emetic on admission. Took ʒvi castor oil in first two days and was very freely purged.	Cured.	About one third of the oil was vomited in this case.
14	W. R—, æt. 40, admitted August 8th. No history.	In extreme collapse; pulse just perceptible; skin livid and very cold; no vomiting or purging; severe cramps; is drowsy and heavy, and has evidently been drinking largely of spirits.	Half-ounce doses of oil given every half-hour until the time of death took place. Enemata and emetics also given.	Died.	Kept gradually sinking and died ten hours after admission.
15	J. L—, æt. 64, admitted August 8th. Was seized immediately after supper last night with vomiting and severe cramps. Was very little purged.	Intense algidæ symptoms; no vomiting or purging; great thirst.	Endeavoured to produce purging with large doses of castor oil, croton oil, talomel, podophylin, &c. Injections of oil, &c., given.	Died.	Was in collapse over two days; was never once purged. Had ʒvij castor oil, 40 grs. calomel, ʒvi croton oil, and 10 grs. of podophylin. Several injections of oil were also given.

No.	History, &c. &c.	Symptoms on admission.	Treatment.	Result.	Remarks.
16	R. M—, æt. 16, admitted August 8th. Was a patient here a week ago with cholera, and was discharged apparently well. Was seized again last night with fresh choleraic symptoms.	Slight collapse; vomiting and pulse very faint and quick.	Had ʒij of castor oil during first day and half. Was very freely purged.	Cured.	This relapse was most likely caused by some irregularity or excess when the girl returned to her home. It is a curious fact that this girl was again admitted with a third attack on the 1st November.
17	J. O—, æt. 10, admitted August 10th. Yesterday after dinner, at which he partook freely of vegetables, he was seized with vomiting, purging, and cramps. Was treated by a medical man.	Considerable collapse; pulse very weak; skin blue and cold; fingers almost black, and very painful to the touch. The extremities of the fingers were gangrenous, and covered with small white blisters.	Free action of the bowels kept up for three days. The fingers (which were wrapped in the dry cotton wool) gradually regained their usual appearance. The boy after being declared convalescent, was ordered liniment for a squamous eruption on the body.	Cured.	There is little doubt that this case would have resulted in gangrene (like one previously reported) had not the circulation rapidly gained power after the purging. Convalescent on fifth day.
18	J. P—, æt. 28, admitted August 11th. Is of very intemperate habits, and has been drinking very hard lately. Has been ill with diarrhœa for some days. Took some medicine. Became much worse this morning.	In a state of great collapse. Pulse just perceptible; algidæ symptoms very intense.	Had ʒij castor oil in first three hours, which did not produce purging. Four drops of croton oil every hour for two doses. This was followed by very free purging. The skin began to get warmer, and all the symptoms less severe.	Cured.	As the oil which was administered at first did not produce purging, and as the patient was rapidly sinking, it was deemed expedient to resort to a stronger purgative. The result is shown, in that the patient was pronounced convalescent on the fourth day.

19	E. F—, æt. 25, admitted August 12th. The patient, being unable to speak, we could obtain no particulars at the time of admission.	In great distress; tongue enormously swollen. Was to be followed in half-an-hour purged twice in transit to the hospital. Very like a case of irritant poisoning.	Ordered calomel five grains, to be followed in half-an-hour by ʒss of castor oil. She had three doses of oil, and was freely purged.	Cured.	The day following her admission, the tongue was so much swollen that the mouth could not be opened. Eightleeches were applied under the jaw, which gave great relief. Patient was convalescent next day.
20	H. N—, æt. 19, admitted August 12th; a vagrant. Had no food yesterday, and was wandering about the streets all night. Was suddenly seized this morning with choleraic symptoms.	Slight collapse; severe vomiting, purging, and cramps.	Had an emetic, four ʒss doses of castor oil, and small quantities of ammonia mixture.	Cured.	Was very freely purged during first day. Convalescent on third day.
21	B. D—, æt. 37, admitted August 13th. Is of phthisical appearance. Has been ill for some days with diarrhœa. Was seized with severe cramps this morning. Has taken no medicine.	Slight collapse. Intense pulmonary emphysema; great vomiting; coughs a good deal; is cold and clammy. Per- sistent vomiting. Pulse almost imperceptible.	It was impossible to give oil on account of the persistent vomiting; ten doses vomited. Injections of oil and warm gruel were given, and free purging followed. A mixture containing hydrocyanic acid was given to allay the vomiting when it became excessive. Effervescing mixtures were given during reaction.	Cured.	There was considerable difficulty experienced in producing purging. When this was effected, however, the patient began to recover. Reaction was marked by considerable pulmonary congestion and fever, the tendency to which was most likely increased by tubercular deposit in the lungs.
22	J. T—, æt. 15, admitted August 13th. Was suddenly seized with a feeling of faintness when at breakfast. Purg-	Considerable collapse. Severe vomiting; no purging; cramps; intense thirst; skin very cold; pulse almost imperceptible;	Castor oil in ʒij doses was given every half hour until the bowels were freely acted upon.	Cured.	Although only five hours had elapsed from the commencement of the attack till the removal to hospital, the patient on ad-

No.	History, &c. &c.	Symptoms on admission.	Treatment.	Result.	Remarks.
16	R. M—, æt. 16, admitted August 8th. Was a patient here a week ago with cholera, and was discharged apparently well. Was seized again last night with fresh choleraic symptoms.	Slight collapse; vomiting and purging a little; pulse very weak and quick.	Had $\mathfrak{ss}$ of castor oil during first day and half. Was very freely purged.	Cured.	This relapse was most likely caused by some irregularity or excess when the girl returned to her home. It is a curious fact that this girl was again admitted with a third attack on the 1st November.
17	J. O—, æt. 10, admitted August 10th. Yesterday after dinner, at which he partook freely of vegetables, he was seized with vomiting, purging, and cramps. Was treated by a medical man.	Considerable collapse; pulse very weak; skin blue and cold; hands and wrists almost black. The extremities of the fingers were gangrenous, and covered with small white blisters.	Free action of the bowels kept up for three days. The fingers (which were wrapped in dry cotton wool) gradually regained their usual appearance. The boy after being declared convalescent, was ordered lime-juice for a squamous eruption on the body.	Cured.	There is little doubt that this case would have resulted in gangrene (like one previously reported) had not the circulation rapidly gained power after the purging. Convalescent on fifth day.
18	J. P—, æt. 28, admitted August 11th. Is of very intemperate habits, and has been ill with diarrhœa for some days. Took some medicine. Became much worse this morning.	In a state of great collapse. Pulse just perceptible; algidæ symptoms very intense.	Had $\mathfrak{ss}$ castor oil in first three hours, which did not produce purging. Four drops of croton oil every hour for two doses. This was followed by very free purging. The skin began to get warmer, and all the symptoms less severe.	Cured.	As the oil which was administered at first did not produce purging, and as the patient was rapidly sinking, it was deemed expedient to resort to a stronger purgative. The result is shown in that the patient was pronounced convalescent on the fourth day.

27	and cramps. Had several doses of chalk mixture. M. K—, æt. 27, admitted August 18th. Was seized this morning with severe cramps, which were soon followed by severe vomiting and purging.	Severe cramps; incessant vomiting; purging of thin flocculent matter.	Had four 3ss doses of castor oil in the first twenty-four hours.	Cured.	Discharged on the fourth day.
28	J. S—, æt. 60, admitted August 19th. Has had diarrhœa for two days. Vomiting and cramps came on this morning.	"Choleroïd diarrhœa."	Had two 3ss doses of castor oil, and was pretty freely purged.	Cured.	Discharged on the fourth day.
29	M. O—, æt. 38, admitted August 20th. One of her children died of cholera yesterday. There is much cholera in the street where she resided. She was seized suddenly this morning with choleraic symptoms.	Severe cramps; persistent vomiting; slight purging; no collapse.	Ordered <i>Spts. Chloroformi</i> co.; this was given up in the evening and castor oil in half-ounce doses given every hour until free purging was produced.	Cured.	Was convalescent on the second day, and discharged on the fourth. The mixture contained <i>Spts. Chloroform. Mist. Cretæ co., Liq. Opii sed.</i>
30	M. M'N—, æt. 23, admitted August 20th; a prostitute. Had been ill since the day before yesterday, when she was seized with vomiting, purging, and cramps. Took a good deal of medicine which she purchased at a chemist's shop.	Is in collapse; is fearfully filthy; skin very cold. Pulse scarcely perceptible.	Had frequent doses of oil up to second day, and was moderately purged. Passed water freely every day.	Died.	Had a strong epileptiform fit on the evening of the second day, shortly after which she died.

No.	History. &c. &c.	Symptoms on admission.	Treatment.	Result.	Remarks.
31	E. C—, æt. (cir.) 48, admitted August 23rd. No history.	Is in complete pulseless collapse.	Had eight ʒss doses of oil and turpentine, and several injections of oil and warm gruel.	Died.	Death took place thirteen hours after admission, during which time purging never once occurred.
32	J. Mc—, æt. 15, admitted August 23rd. Was suddenly taken ill a short time ago with vomiting, purging, and cramps.	"Choleroïd diarrhœa."	Had four ʒijj doses of castor oil; was freely purged.	Cured.	Discharged on the third day.
33	M. C—, æt. 37, admitted August 24th; a tailor. Is addicted to drinking. Was suddenly seized last night with severe cramps, followed by purging and vomiting. Took several doses of a medicine obtained at the Richmond Row Dispensary.	Persistent vomiting; little pulse; no cramps; pupils contracted; ex-castor oil.	Had an emetic; free purgation produced and kept up by ex-castor oil.	Cured.	Had about four ounces of oil, and was purged nineteen times. Convalescent on the third day.
34	J. R—, æt. 28, admitted August 25th; a sailor. Took ill with vomiting and purging last night. Was seized suddenly, and felt very cold at the time.	Vomiting persistent. Purged while being undressed; pulse cold; extremities very weak; eyeballs much sunken.	Had three ʒj doses of castor oil during the first day. The second day he had ʒj of oil, after which he was purged five times. Third day had no oil, but was freely purged.	Cured.	On the evening of the third day he had a very severe ague fit which left him very weak. He was then ordered <i>Mist. Quinæ fort.</i>

35	E. C—, æt. 24, admitted August 26th; a patient in the cleansing ward of the work-house. Was eating "scoces" for supper last night; was seized with vomiting, purging, and cramps this morning.	Is in slight collapse; vomiting a little; pulse very weak; skin cold; great thirst.	To have $\frac{3j}{\text{of}}$ castor oil. Heat, dinapiams, &c. Oil to be repeated in two hours. To have ammonia mixture every hour. Had $\frac{3j}{\text{of}}$ oil during second day.	Cured.	Had about three ounces of oil; was very freely purged and declared convalescent on the third day. Reaction was marked by slight fever.
36	E. C—, æt. 23, admitted August 26th. Has had diarrhoea for two days. Took medicine.	Is in slight collapse; vomiting very severe; pulse very weak; temperature much reduced.	Had an emetic. Had three $\frac{3j}{\text{of}}$ doses of castor oil during first day; was freely purged. Oil given every day until the day of discharge.	Cured.	Convalescent on the fourth day.
37	R. T—, æt. 71, admitted August 26th. Has had diarrhoea for two days. Was seized with vomiting and cramps about an hour since.	Slight collapse; sunken eyeballs; livid, cold extremities; respiratory embarrassment; severe cramps; persistent hiccup.	Had three $\frac{3j}{\text{of}}$ doses of castor oil, and two oil and gruel injections.	Cured.	Was very freely purged during the first day and night. Discharged on the third day.
38	M. A. S—, æt. 46, admitted August 28th. Has had diarrhoea for three days. Vomiting and cramps set in this morning; was taking medicine from one of the public dispensaries.	Severe "choleraic diarrhoea."	Had two $\frac{3j}{\text{of}}$ doses of castor oil and one injection of oil and gruel.	Cured.	Was very freely purged by oil. Convalescent on second day.
39	J. W—, æt. 22, admitted August 28th. Brought from the "Classification Ward" of the workhouse, where she was seized some few hours ago with severe cramps, vomiting, and purging.	Vomiting and purging a good deal; cramps severe; pulse weak and very quick.	Had $\frac{3j}{\text{of}}$ of castor oil in first twelve hours; was very freely purged. Action of oil kept up.	Cured.	Convalescent on the second day.



No.	History, &c. &c.	Symptoms on admission.	Treatment.	Result.	Remarks.
40	M. A. R—, æt. 49, admitted August 28th. Was discharged from the cholera sheds in Ashfield Street to-day, and shortly after leaving was seized with choleraic symptoms. Was brought here by a policeman.	Slight collapse; severe cramps, cold skin, &c.	Had five ʒss doses of oil, and was eight times freely purged.	Cured.	The relapse here may have been due to some excess indulged in by the patient when discharged from the cholera sheds.
41	E. M—, æt. 22, admitted August 30th. Is a convalescent from typhus fever. Was seized this morning in the convalescent fever ward with severe choleraic symptoms.	Considerable collapse; pulse excessively weak; skin cold and clammy.	Had two ʒij doses of castor oil on first day. She was very freely purged, and was then ordered <i>Mist. Rosa</i> .	Cured.	Convalescent on the third day.
42	J. C—, æt. 11, admitted August 30th. Is a very delicate-looking child. Has been ill with diarrhœa for two days. Became much worse this morning.	Vomiting, purging, and cramps; slight collapse.	Had grey powder, and two ʒij doses of oil which produced considerable purgation, and gave great relief.	Cured.	The child had a brisk reactionary fever which lasted four days.
43	E. C—, æt. 26, admitted August 31st. Is a prostitute. Has had diarrhœa for two days. Has been drinking a great deal lately. Took a good deal of medicine obtained at a chemist's.	In extreme collapse; extremities livid; eyes much sunken; pulseless at radial; passed water for a short time since; vomited a few minutes ago; no purging; no cramps; respiration	Ordered ʒvj castor oil and ʒij turpentine every two hours for three doses. These were retained and followed by free purging. Second day had ʒss castor-oil. Third day had ʒss	Cured.	She had about eight ounces of oil and was purged very freely and frequently during the first two days. Reaction was marked by considerable feverishness, which, however, soon subsided.

	embarrassed.	castor oil. Small quantities of arrowroot, &c., were given on second and third days.	Convalescent on fifth day.
44 H. W—, æt. 20, admitted September 1st. Is a vagrant. Was suddenly seized in the Vagrant Sheds to-night with severe choleraic symptoms.	Severe "choleroïd diarrhœa," with slight collapse.	Had three 3ss doses of castor oil, and was purged moderately in first forty-eight hours. Placed on strong quinine mixture on the third day.	Convalescent and put on full diet third day. Discharged sixth day.
45 W. I—, æt. 47, admitted September 1st. Brought from the Royal Infirmary at 7 a.m. Was seized in the Infirmary (where he was a patient) with choleroïd symptoms this morning.	Considerable collapse. Pulse barely perceptible; skin very cold and clammy; greatly emaciated; extremities livid; intense thirst, and severe cramps.	Had in the thirteen hours which he was in hospital eight ounces of castor-oil and was purged about twenty times. He sank from intestinal hæmorrhage probably.	The last half-dozen stools contained a considerable quantity of blood. There was probably a great deal too much oil given in this case, considering the stamina of the patient.
46 E. D—, æt. 42, admitted September 1st. Was taken ill yesterday with vomiting, purging, and cramps. Was "on the spree" the day before.	Pulseless collapse; severe cramps.	Had two 3ss doses of castor oil and three of the potash electuary. Two drops of croton oil were rubbed on tongue, and four oil and gruel injections given.	Effort after effort was made to produce purgation, but in vain. The patient was never purged until in <i>articulo mortis</i> . Died nine hours after admission.
47 M. D—, æt. 26, admitted September 1st. Was brought from the Classification Wards of the workhouse where she was seized last night with choleroïd symptoms.	"Choleroïd diarrhœa," with slight collapse.	Had three doses of oil in first six hours which produced very free purgation. A strong astringent mixture was then administered, shortly after which the patient became feverish. Elimination, however, con-	This case affords an excellent illustration of "the natural mode of recovery," and of the results of "waging an intestine war." Convalescent on sixth day.

No.	History, &c. &c.	Symptoms on admission.	Treatment.	Result.	Remarks.
48	M. R—, set. (cir.) 30, admitted September 2nd. Was seized this morning with choleric symptoms. Is of intemperate habits; mother believes she was drinking yesterday.	In pulseless collapse; unable to speak; algid symptoms very intense; great pulmonary emphysema; great pulmonary embarrassment.	continued, the patient being more or less purged every day until her discharge. Friction with oil of mustard. Dry heat, &c. Had $\frac{3}{4}$ j castor oil by mouth and several ounces by injection. Was purged once scantily.	Died.	This patient died about six hours after admission, having been purged only once. Vomiting never occurred.
49	F. B—, set. 34, admitted September 3rd. Was seized about an hour ago with severe choleric symptoms. Is of temperate habits.	"Choleric diarrhœa."	Had three $\frac{3}{4}$ s doses of castor oil with $\frac{3}{4}$ j of turpentine in each.	Cured.	Was very freely purged. Convalescent on the second day.
50	D. E—, set. 46, admitted September 4th. Is of very intemperate habits and was drinking very hard all last week. Was seized with vomiting and purging this morning. Took nothing but some hot gin.	"Choleric diarrhœa."	Ordered $\frac{3}{4}$ j of castor oil on admission, and another in two hours.	Cured.	Had $\frac{3}{4}$ j of castor oil; was purged very freely and pronounced convalescent on second day.
51	—, apparently about 26 years of age, admitted September 6th, a Swede. Took ill on	Is in complete pulseless collapse.	Ordered $\frac{3}{4}$ s of castor oil, and $\frac{3}{4}$ j turpentine every hour. Hot injections to be given	Died.	Retained six doses of oil. Was scantily purged after the injections. Died fourteen hours

52	<p>his way from Sweden with vomiting, purging, and cramps.</p> <p>E. H—, æt. 25, admitted September 6th. Came from Hamburg a few days ago. Has been ill with diarrhœa for some days. Has been getting worse for last twenty-four hours.</p>	<p>In a state of extreme collapse.</p>	<p>every hour. Ammonia mixture. Friction with oil of mustard. Dry heat, &amp;c.</p> <p>3vj of castor oil given at once. Was repeated in forty-five minutes. This did not produce purging; oil to be repeated every hour.</p>	<p>Died.</p>	<p>Had five doses of oil and several injections of oil and warm gruel. Died six hours after admission, having never been purged.</p>	<p>after admission.</p>
53	<p>W. T—, æt. 16, admitted September 7th. Has had diarrhœa for two days. Was seized with vomiting, purging, and cramps in the Vagrant Sheds this morning.</p>	<p>Severe diarrhœa.</p>	<p>Ordered 3ss of oil every hour for four doses; second day had 3j of oil.</p>	<p>Cured.</p>	<p>Had 3ij of castor oil; was freely purged; convalescent on second day.</p>	
54	<p>W. T—, æt. 16, admitted September 7th. Was suddenly seized about twelve hours ago with severe cramps and profuse purging. Had been eating largely of mussels.</p>	<p>Very severe "choleroïd diarrhœa."</p>	<p>Had three 3ij doses of castor oil with ten drops of chlorodyne in each. Free purgation with great relief followed. Convalescent on second day.</p>	<p>Cured.</p>	<p>The diarrhœa was most probably caused by the mussels eaten. (It is well known that the whole family (<i>Mytilaceæ</i>) are poisonous at certain seasons and when taken from particular beds. J. W. McC.)</p>	
55	<p>E. D—, æt. 34, admitted September 7th. Is of intemperate habits. Has been ill since yesterday morning, when he was suddenly seized with choleroïd symptoms.</p>	<p>Considerable collapse; severe cramps; vomiting a little; no purging; algidæ symptoms well marked.</p>	<p>During first three days had 3iv of oil, and was very freely purged. Was declared convalescent on the fourth day and ordered full diet.</p>	<p>Cured.</p>	<p>On the evening of the fourth day he had a slight relapse; oil was again administered and he soon recovered.</p>	

No.	History, &c. &c.	Symptoms on admission.	Treatment.	Result.	Remarks.
56	J. R—, æt. 43, admitted September 9th. Was seized yesterday morning with profuse purging. He vomited very much shortly after, and the diarrhœa continued until about four hours ago.	Pulse small and very weak; skin cold and clammy; eyeballs sunken; respiration a little embarrassed.	3ss of castor-oil was given on admission; about half an afterwards he passed a bloody stool. Injections of starch and opium were then administered, and "Rose mixture" ordered.	Cured.	He made a rapid recovery, but was kept in hospital a week or so.
57	R. L—, æt. 56, admitted September 10th. Has had diarrhœa for four or five days, accompanied by severe cramps. He became worse this morning. Took some medicine.	Severe diarrhœa.	Had five doses of oil, the first action of which brought a quantity of scybala from the intestinal canal.	Cured.	Was convalescent on the second day. The scybala were undoubtedly the cause of the diarrhœa in this case.
58	W. C—, æt. 52, admitted September 10th. Is a fat man and works at the Queen's dock. There is no history.	In pulseless collapse; is unable to speak; pulse only just perceptible in brachial artery.	Large quantities of oil given by mouth and by injections.	Died.	Death took place ten hours after admission. The patient was never once purged.
59	W. E—, æt. 28, admitted September 13th. Was brought from Garston. Is of very intemperate habits. Was seized with choleric symptoms this morning. Took great quantities of brandy.	Has a wild expression; is apparently drunk; furious delirium; legs much cramped; no purging or vomiting; pulse almost imperceptible; algidæ symptoms intense; great dyspnoea.	Mustard emetic given followed by a dose of oil and turpentine every hour.	Died.	Death took place about twelve hours after admission. Scarcely any vomiting and no purging while in hospital.

60	M. M—, set. 48, admitted September 13th. Is of in-temperate habits. Was seized at 5 o'clock this morning with severe cramps, which were soon followed by an attack of profuse purging. Was not ailing yesterday.	In slight collapse; severe cramps and intense thirst; pulse very quick and feeble; algidæ; symptoms well-marked.	3ss of castor oil and ℥j of turpentine every second hour. Free purging was produced. Was convalescent on third day. Action of bowels kept up until the day of discharge.	Cured. Had altogether about 3iv of oil. Was very freely purged first day.
61	J. P—, set. 40, admitted September 14th. A vagrant from the Vagrant Sheds. Had diarrhœa all day yesterday; was suddenly seized with vomiting and cramps during the night.	Persistent vomiting; slight purging; approaching collapse; temperature sensibly reduced.	A mustard emetic. Had two doses of castor oil and turpentine on first day.	Cured. Was very freely purged. Convalescent on second day. Action of bowels kept up until day of discharge.
62	J. S—, set. 48, admitted September 15th. Is of very in-temperate habits. Has been ill for two days with diarrhœa. Was seized to-day with vomiting and cramps.	In considerable collapse; pulse just perceptible at radial. No purging or vomiting; passed water a short time ago.	3ss doses of castor oil with ℥j of turpentine, and ℥xv of chlorodyne given every hour. Small quantities of ammonia and brandy given.	Died. This patient remained in collapse about sixteen hours, during which time he was only once purged.
63	J. R—, set. 8, admitted September 17th. A delicate strumous-looking child. Has had diarrhœa for a few days, this was accompanied by griping pains. Was seized with vomiting this morning, when the purging also became worse.	"Choleroid diarrhœa."	Had five powders containing six grains of grey powder each. Was very freely purged for some days. Starch enemata were subsequently given. Small quantities of wine and arrow-root given from the commencement.	Cured. This was most likely a neglected case of irritative diarrhœa. The evacuations contained much undigested food &c.

No.	History, &c. &c.	Symptoms on admission.	Treatment.	Result.	Remarks.
56	J. R—, æt. 43, admitted September 9th. Was seized yesterday morning with profuse purging. He vomited very much shortly after, and the diarrhœa continued until about four hours ago.	Pulse small and very weak; skin cold and clammy; eyeballs sunken; respiration a little embarrassed.	3ss of castor-oil was given on admission; about half an afterwards he passed a bloody stool. Injections of starch and opium were then administered, and "Rose mixture" ordered.	Cured.	He made a rapid recovery, but was kept in hospital a week or so.
57	R. L—, æt. 56, admitted September 10th. Has had diarrhœa for four or five days, accompanied by severe cramps. He became worse this morning. Took some medicine.	Severe diarrhœa.	Had five doses of oil, the first action of which brought a quantity of scybala from the intestinal canal.	Cured.	Was convalescent on the second day. The scybala were undoubtedly the cause of the diarrhœa in this case.
58	W. C—, æt. 52, admitted September 10th. Is a fat man and works at the Queen's dock. There is no history.	In pulseless collapse; is unable to speak; pulse only just perceptible in brachial artery.	Large quantities of oil given by mouth and by injections.	Died.	Death took place ten hours after admission. The patient was never once purged.
59	W. E—, æt. 28, admitted September 13th. Was brought from Garston. Is of very intemperate habits. Was seized with choleraic symptoms this morning. Took great quantities of brandy.	Has a wild expression; is apparently drunk; furious delirium; legs much cramped; no purging or vomiting; pulse almost imperceptible; algidæ symptoms intense; great dyspnoea.	Mustard emetic given followed by a dose of oil and turpentine every hour.	Died.	Death took place about twelve hours after admission. Scarcely any vomiting and no purging while in hospital.

60	M. M—, æt. 48, admitted September 13th. Is of in-temperate habits. Was seized at 5 o'clock this morning with severe cramps, which were soon followed by an attack of profuse purging. Was not ailing yesterday.	In slight collapse; severe cramps and intense thirst; pulse very quick and feeble; algidæ; symptoms well-marked.	3ss of castor oil and ʒj of turpentine every second hour. Free purging was produced. Was convalescent on third day. Action of bowels kept up until the day of discharge.	Cured. Had altogether about ʒiv of oil. Was very freely purged first day.
61	J. P—, æt. 40, admitted September 14th. A vagrant from the Vagrant Sheds. Had diarrhœa all day yesterday; was suddenly seized with vomiting and cramps during the night.	Persistent vomiting; slight purging; approaching collapse; temperature sensibly reduced.	A mustard emetic. Had two doses of castor oil and turpentine on first day.	Cured. Was very freely purged. Convalescent on second day. Action of bowels kept up until day of discharge.
62	J. S—, æt. 48, admitted September 15th. Is of very in-temperate habits. Has been ill for two days with diarrhœa. Was seized to-day with vomiting and cramps.	In considerable collapse; pulse just perceptible at radial. ʒij of turpentine, and ℥xv of chlorodyne given every hour. No purging or vomiting; passed small quantities of ammonia and water a short time ago. brandy given.	3ss doses of castor oil with ʒij of turpentine, and ℥xv of chlorodyne given every hour. Small quantities of ammonia and brandy given.	Died. This patient remained in collapse about sixteen hours, during which time he was only once purged.
63	J. R—, æt. 8, admitted September 17th. A delicate strumous-looking child. Has had diarrhœa for a few days, this was accompanied by griping pains. Was seized with vomiting this morning, when the purging also became worse.	"Choleroïd diarrhœa."	Had five powders containing six grains of grey powder each. Was very freely purged for some days. Starch enemata were subsequently given. Small quantities of wine and arrow-root given from the commencement.	Cured. This was most likely a neglected case of irritative diarrhœa. The evacuations contained much undigested food &c.



No.	History, &c. &c.	Symptoms on admission.	Treatment.	Result.	Remarks.
64	M. M'C—, æt. 5, admitted September 17th. Was brought from the "Emigrant Sheds" where her parents died of cholera. Had diarrhœa all day yesterday.	Considerable collapse; pulse just perceptible; skin very cold; eyeballs much sunken.	Had small doses of castor oil. Body rubbed with oil.	Died.	The oil was given up till the time of death (nine hours after admission). Was purged twice very scantily; never vomited.
65	S. B—, æt. 18, admitted September 17th. Was picked up from the streets by a policeman. Was seized about five hours ago with choleroïd symptoms.	"Choleroïd diarrhœa."	Free action of bowels produced and kept up by oil and turpentine. <i>Mist. Potass. Chlor.</i> given during the reactionary fever.	Cured.	Was rather feverish on second day. Convalescent on third day.
66	C. O'G—, æt. 32, admitted September 18th. A vagrant, admitted from the Vagrant Sheds, where she was seized about two hours ago with vomiting, purging and cramps.	"Choleroïd diarrhœa."	Had six ʒss doses of castor oil in first thirty-six hours. Was freely purged.	Cured.	Discharged on the third day.
67	S. F—, æt. 61, admitted September 20th. Had diarrhœa for a week. Was suddenly seized with vomiting and cramps yesterday evening. Took some medicine.	Great collapse; pulse very feeble; cramps very severe; skin cold and clammy; considerable dyspnoea.	ʒss doses of oil and turpentine given every hour up till the time of death. Injections of oil were also given but were not retained.	Died.	Remained in collapse for twelve hours, during which time she was never once purged.

68	J. J—, æt. 44, admitted September 22nd, a vagrant. Brought from the Vagrant Sheds where he was suddenly seized with choleric symptoms.	Slight collapse; skin cold; pulse very weak; vomiting a little; complains of cramps.	Free action of bowels kept up with oil and turpentine for three days.	Convalescent, and put on full diet on the third day.
69	L. N—, æt. 35, admitted September 23rd, had diarrhoea for two days. Was treated at one of the dispensaries. Got worse this morning.	No vomiting or purging; collapse rapidly setting in; pulse just perceptible at wrist.	Had 3ss of oil every hour with ʒj of turpentine, ʒj of brandy ordered every second hour, and a little ammonia mixture frequently.	Collapse rapidly set in, from which she never once rallied. Was purged three times. Died on the third day.
70	A. I. I—, æt. 26, admitted September 25th. Is a prostitute. Has been ill for two days with diarrhoea. Vomiting began last night. Took some brandy and laudanum.	Almost pulseless; algidæ symptoms well-developed. No purging or vomiting.	Treatment commenced with gr. vj. <i>Pulv. Opii</i> gr. ʒ. <i>Saccharum</i> gr. vj. Oil in ʒss doses every hour was given soon afterwards and continued for four doses. Free purging was produced and the patient was convalescent on the second day.	This case is a good illustration of the rapidity with which patients recover from cholera collapse.
71	S. P—, æt. 60, admitted September 28th. Was a patient in the surgical wards of the Infirmary with uterine disease. Was very suddenly seized with choleric symptoms this morning. Was immediately transferred to the Cholera-reserve Ward.	Notwithstanding the short time that has elapsed since the commencement of the attack, the patient is now in a state of extreme collapse; pulse is almost imperceptible and there is considerable pulmonary embarrasment.	Ordered powders containing <i>Calomel</i> gr. vj, one every hour. These were retained but did not produce purging, although enemas of castor oil were at the same time administered.	Death took place about nine hours after admission. The difficulty in producing purging here was probably due to the small stream of blood issuing from the lungs, as evidenced by the dyspnoea and feeble pulse.

No.	History, &c. &c.	Symptoms on admission.	Treatment.	Result.	Remarks.
72	M. J. W—, æt. 18, admitted September 28th. A pauper from the Receiving-house of the Workhouse. Was seized last night with diarrhœa, and this morning with vomiting and cramps.	Vomiting a little; no purging; severe cramps; pulse very weak.	Had an emetic, followed by 3ss of castor oil and 3ij of turpentine every hour, for three doses.	Cured.	Had slight reactionary fever. Convalescent on the fourth day.
73	J. C—, æt. 49, admitted October 1st. A pauper brought from the female "Labour Test" department. Was suddenly seized with choleroïd symptoms about three hours ago. Got no medicine.	Vomiting a little; no purging; pulse very weak; skin cold and clammy.	Had powders, containing <i>Calomelana</i> . gr. x, Pulv. Opii gr. ½. One every hour. Took four.	Cured.	Was very freely purged. Convalescent on the fourth day.
74	E. F—, æt. 21, admitted October 1st. Was seized at noon to-day with vomiting, purging, and severe cramps in stomach.	Severe "choleroïd diarrhœa."	Had an emetic. Free purging produced with castor oil and turpentine.	Cured.	Convalescent on the third day.
75	M. F—, æt. 53, admitted October 2nd. Has had diarrhœa for some days, choleroïd symptoms set in this morning.	"Choleroïd diarrhœa" approaching collapse.	Had an emetic, ʒiiss of castor oil and three oil and gruel injections.	Cured.	Very freely purged; convalescent on the sixth day.

76	M. F—, æt. 53, admitted October 2nd. Has had diarrhœa for a few days, vomiting, skin cold; complains of severe cramps began this morning.	Slight collapse; no vomiting or purging; pulse very weak; skin cold; complains of severe cramps.	Had one or two doses of castor oil and three powders of calomel (gr. v). Ammonia mixture was given every hour in small quantities.	Cured. Purged very freely; convalescent on the fourth day.
77	C. K—, admitted October 3rd. No history.	In considerable collapse; great thirst; persistent vomiting and very marked sinking of the eyeballs.	Small doses of oil were given but immediately rejected. Grey powder and calomel shared a like fate.	Died. Death occurred about twenty-three hours after admission. Was never once purged, but vomited incessantly.
78	J. J—, æt. 23, admitted October 4th. A vagrant from the Vagrant Sheds. Has had diarrhœa for a few days. Was seized a few hours ago with vomiting and cramps.	Severe diarrhœa.	Had an emetic and $\frac{3}{4}$ ij of castor oil on first day. Was freely purged.	Cured. Discharged on the second day.
79	M. M—, æt. 43, admitted October 6th. Has had diarrhœa for ten days, was taking medicine from one of the dispensaries. Felt much worse this afternoon.	"Choleroïd diarrhœa."	Had $\frac{3}{4}$ ij of castor oil and was very freely purged. Small quantities of beef-tea, arrowroot and wine given every second hour.	Cured. The purging here was very severe. The last stools were bloody. Starch and opium enemata were then given. Convalescent on the fifth day.
80	A. J—, æt. 47, admitted October 8th. Was brought from the Surgical Wards, where she was being treated for a large phagedenic ulcer of the leg. Has been ill for years and is greatly emaciated.	In a state of collapse; pulse almost imperceptible; vomiting and purging.	Ordered $\frac{3}{4}$ s of castor oil after which she was purged twice. She did not rally, however, but continued sinking in collapse, and died about six hours after admission.	Died. It was not to be wondered at that this patient, suffering for such a length of time from an exhaustive disease, should succumb so soon to cholera.

No.	History, &c. &c.	Symptoms on admission.	Treatment.	Result.	Remarks.
	seized during the night with severe cramps, which were soon followed by a profuse diarrhœa.				
81	W. M—, æt. 37, admitted October 9th. A vagrant from the Vagrant Sheds. Has been ill for a few days with diarrhœa. Became much worse a few hours ago.	Severe diarrhœa.	Had a few doses of castor oil and turpentine during first day. Put on <i>Mist. Quinæ fort.</i> on second day.	Cured.	Was freely purged. Convalescent on the second day.
82	J. W—, æt. 48, admitted October 9th. Is just out of prison, where he had fever. Was seized with choleraïd symptoms this morning.	Is in pulseless collapse.	Had six 3ss doses of castor oil with ʒi turpentine in each at half-hour intervals. Ammonia mixture was given every hour in ʒss doses.	Died.	This was manifestly a very bad subject for cholera. He seemed moribund from the first moment. He was purged twice very scantily, and died a few hours after admission.
83	S. E—, æt. 24, admitted October 11th. Had an attack of cholera about a week ago, from which she was treated at one of the dispensaries. Has not been exactly well since. Choleraïd symptoms came on this morning.	Slight collapse; skin cold and clammy; pulse very weak; eyes closed; much sunken; severe cramps, &c.	3ss doses of castor oil every hour for four hours, with injections of castor oil. Small quantities of ammonia given. Oil had to be repeated for two more doses before free purgation was produced.	Cured.	Had ʒiij of oil and three injections of oil (ʒi). She was freely purged. Her stamina may be judged of by the fact that she had bed sores on the fourth day.

76	M. F—, æt. 53, admitted October 2nd. Has had diarrhœa for a few days, vomiting, skin cold; complains of severe cramps and cramps began this morning.	Slight collapse; no vomiting; or purging; pulse very weak (gr. v). Ammonia mixture was given every hour in small quantities.	Had one or two doses of castor oil and three powders of calomel (gr. v). Ammonia mixture was given every hour in small quantities.	Cured. Purged very freely; convalescent on the fourth day.
77	C. K—, admitted October 3rd. No history.	In considerable collapse; great thirst; persistent vomiting and very marked sinking of the eyeballs.	Small doses of oil were given but immediately rejected. Grey powder and calomel shared a like fate.	Died. Death occurred about twenty-three hours after admission. Was never once purged, but vomited incessantly.
78	J. J—, æt. 23, admitted October 4th. A vagrant from the Vagrant Sheds. Has had diarrhœa for a few days. Was seized a few hours ago with vomiting and cramps.	Severe diarrhœa.	Had an emetic and ʒij of castor oil on first day. Was freely purged.	Cured. Discharged on the second day.
79	M. M—, æt. 43, admitted October 6th. Has had diarrhœa for ten days, was taking medicine from one of the dispensaries. Felt much worse this afternoon.	"Choleroïd diarrhœa."	Had ʒij of castor oil and was very freely purged. Small quantities of beef-tea, arrowroot and wine given every second hour.	Cured. The purging here was very severe. The last stools were bloody. Starch and opium enemas were then given. Convalescent on the fifth day.
80	A. J—, æt. 47, admitted October 8th. Was brought from the Surgical Wards, where she was being treated for a large phagedenic ulcer of the leg. Has been ill for years and is greatly emaciated. Was	In a state of collapse; pulse almost imperceptible; vomiting and purging.	Ordered ʒas of castor oil after which she was purged twice. She did not rally, however, but continued sinking in collapse, and died about six hours after admission.	Died. It was not to be wondered at that this patient, suffering for such a length of time from an exhaustive disease, should succumb so soon to cholera.

No.	History, &c. &c.	Symptoms on admission.	Treatment	Result.	Remarks.
88	W. O—, æt. 54, admitted October 16th. Has had diarrhœa for two days. Took a good deal of gin. Became much worse this morning.	"Choleroïd diarrhœa," with slight collapse.	Had four ʒss doses of castor oil with ʒj turpentine in each.	Cured.	Was very freely purged. Convalescent on the third day.
89	D. C—, æt. 56, admitted October 17th. Has been ill with diarrhœa "off and on for three weeks." Became much worse this morning.	"Choleroïd diarrhœa."	Treatment commenced with <i>Tinct. Opil</i> and <i>Infus. Ilemacostyl</i> . This was soon discontinued and castor oil in ʒss doses given every hour and half. Very free purging followed the third dose. He, however, convalesced rapidly.	Cured.	On the third day he eat heartily of pea soup and was shortly afterwards seized with choleroïd symptoms for the second time. He recovered under purgation and was discharged on the eleventh day.
90	C. D—, æt. 40, admitted October 18th. Was taken ill this morning with vomiting, purging, and cramps. Is of intemperate habits, but was not drinking yesterday.	Approaching collapse; great thirst; severe cramps; husky voice; pulse very quick and feeble; eyeballs sunken.	ʒss castor oil every hour for three doses. A little ammonia mixture every hour. Small quantities of arrowroot, wine, &c., given on second day.	Cured.	This patient was convalescent and put on quinine mixture the second day. Was freely purged during the first two days. Action of bowels ensured daily until the day of discharge.
91	W. H—, æt. 22, admitted October 22nd. Is of intemperate habits. Has been ill for many days with diarrhœa. Vomiting and cramps set in this morning.	"Choleroïd diarrhœa," with slight collapse.	Had six ʒss doses of castor oil with ʒj turpentine in each. A little ammonia mixture was given every hour.	Cured.	Was freely purged, and was convalescent on the third day.

92	B. R—, æt. 16, admitted October 22nd. Has had diarrhœa for a week, for which he took a good deal of medicine. Became much worse this morning.	"Choleroïd diarrhœa."	Had about $\frac{3}{4}$ of castor oil in first two days and was freely purged.	Cured.	Convalescent on the third day.
93	A. B—, æt. 46, admitted October 22nd. A nurse in the measles ward of the workhouse. Was suddenly seized a short time ago with choleroïd symptoms.	Severe cramps; vomiting and slight purging; pulse very weak; extremities icy cold.	Had $\frac{3}{4}$ of castor oil and three $\frac{3}{4}$ injections of oil on first day.	Cured.	Convalescent on the second day.
94	A. R—, æt. 39, admitted October 30th. Was eating a pork dinner yesterday, soon after which she was seized with vomiting and purging. This was soon followed by severe cramps in the stomach, legs, and arms.	Considerable collapse; skin cold and clammy; pulse very quick and weak; severe cramps; 15 minims of chlorodyne. Both doses were retained but neither produced purging. Next morning two more doses of oil given. Very profuse purging followed.	Ordered $\frac{3}{4}$ ss castor oil, to be given again in half an hour with 15 minims of chlorodyne. Both doses were retained but neither produced purging. Next morning two more doses of oil given. Very profuse purging followed.	Cured.	This patient had only about two ounces of oil, yet she was so freely purged that it was considered advisable to administer enemata of starch and laudanum. Convalescent on third day.
95	R. M—, æt. 16, admitted November 1st. Has been in the "receiving house" of the workhouse for some days. Was suddenly seized with vomiting and purging this morning.	Slight collapse; no vomiting or purging; pulse very weak; skin cold; severe cramps.	Had three $\frac{3}{4}$ ss doses of oil and three calomel powders (5 gr.). Small quantities of ammonia mixture were given every hour.	Cured.	Purged very freely. Convalescent on the fourth day.



No.	History, &c. &c.	Symptoms on admission.	Treatment.	Result.	Remarks.
96	A. B—, æt. 27, admitted November 1st. Was suddenly seized this morning with severe purging and cramps, accompanied by a sense of faintness and sinking. Took some medicine obtained at dispensary.	Almost pulseless; tongue and breath icy cold; voice husky; extremities livid; severe cramps and intense thirst.	Ordered $\frac{3}{4}$ ss castor oil every hour for five times. This produced very free purging during the night. Had 18 grs. calomel and four doses of oil next day, which also produced free purging. About one third of the oil was vomited.	Cured.	Was purged about twenty times. Convalescent on the fourth day.
97	J. C—, æt. 15, admitted November 5th. Was taken ill at twelve o'clock to-day with vomiting, purging, and cramps. Had not been eating anything unusual.	In a state of complete, pulseless collapse.	Ordered $\frac{3}{4}$ ij castor oil with 15 minims chlorodyne, to be repeated again in two hours. A little ammonia mixture every hour. Friction, &c. Had two doses of oil first day, and was purged freely during that night. Ordered injection next day which was immediately returned. Purged twice second day. Still pulseless. Ordered oil again.	Died.	This patient had about two ounces of oil and was not profusely purged. He continued sinking until the evening of the second day, when he passed two stools of blood and water, and died.
98	J. S—, æt. 27, admitted November 7th. Is of moderately temperate habits. Has been ill since yesterday morning with choleric symptoms.	Is in slight collapse; severe cramps; vomiting freely.	An emetic. $\frac{3}{4}$ ss castor oil and $\frac{3}{4}$ ij turpentine every second hour. <i>Acet. Potass</i> ordered on third day.	Cured.	Had four doses of oil; was very freely purged; vomited a great deal. Was convalescent on fourth day.

99	J. P—, æt. 33, admitted November 10th. Was taken ill this morning with choleraïd symptoms. Had taken a pork supper.	"Choleraïd diarrhœa."	Had fourteen half ounce doses of oil in two days.	Cured.	Very free purging produced. Convalescent on the third day.
100	R. T—, æt. 28, admitted November 10th. A labourer, is of intemperate habits. Was seized with profuse purging and vomiting this morning.	Severe "choleraïd diarrhœa."	Had four half-ounce doses of castor oil on first day, 3j of oil on second.	Cured.	Was very freely purged. Discharged on third day.

## DIARRHŒA.

Our experience of diarrhœa was very extensive. Several thousand cases came under our observation in the different dispensaries connected with the West Derby Union and in the Liverpool Parish infirmary. Among these were doubtless many which would have recovered under any mode of treatment or by the *vis medicatrix nature* alone. But there were many too of a most severe choleraic type. The treatment adopted was generally evacuant in its nature and consisted in the administration of castor oil, calomel, rhubarb, or magnesia. In every case relief was afforded "pleasantly, quickly, and safely." It was but seldom that more than two or three doses of oil were required. In one of the public dispensaries (Bootle), in the neighbourhood of Liverpool, many cases of diarrhœa were treated with evacuants and the testimony of the medical officers is in accordance with our own, "We certainly had less trouble with the evacuant mode of treatment. Our patients seldom gave us a third visit, two doses of castor oil or rhubarb mixture being generally sufficient to cure the disease." We found the ordinary fluid magnesia a safe and active aperient in the diarrhœa of infants. We never saw a diarrhœa patient, treated with evacuants from the commencement of his attack, require subsequent removal to hospital.

Upon looking over our cases it will be observed that in a large proportion of them there was "premonitory diarrhœa" which had been treated, often for four or five days, with astringents. Diarrhœa patients undoubtedly recover when treated with astringents, but the recovery is not consequent upon the arrest of the discharges, as these are invariably restored before the patient feels well.

Before coming to any conclusion upon the value of the evacuant treatment of cholera, as based upon our cases, it must be remembered that in nearly every case the patient had been previously drugged with opiates and astringents. Popular "cholera mixtures"—strongly astringent—were

administered in the most indiscriminate manner upon the slightest approach of choleraic symptoms. It was the exception for us to get a patient in the stage of evacuation. There was a strong and very natural antipathy to "the house," amongst the people from whom our patients were mostly taken. They preferred trying the virtue of cholera mixtures and brandy for a considerable time before consenting to removal. We have the concurrent testimony of many of the medical officers in Liverpool and of the parish relieving officers, to the effect that "it was the last shift, when they could induce the friends of a cholera patient, or the patient himself to consent to his removal to the Workhouse."

We consider these remarks necessary for reasons which shall appear hereafter.

*General results of post-mortem examinations*—Nine *post-mortem* examinations were made. The appearances found, upon examining patients who had died during collapse, were wonderfully similar, so far as the organs of circulation and respiration were concerned. The right side of the heart, the pulmonary artery and its larger divisions were invariably distended with thick dark blood, while the ultimate lung tissue was pale and exsanguineous. We regret to say, that in no case was the *weight* of the lungs determined. Only one examination was made of a patient who had died during the period of reaction; and on reference to this case it will be observed, that there was congestion and purulent infiltration of the lungs. The patient during life exhibited marked symptoms of pneumonia. This case leads us to make a few observations upon the

*Period of reaction*.—The tendency to accidents and complications during this period was very great. Pneumonia was a very common and often rapidly fatal complication. There is a natural tendency to pulmonary congestion during reaction, and it is very easily increased. Exposure to draughts of cold air, and the injudicious administration of nitrogenous food, contributed largely, we believe, to the production of this complication in our cases. *Uræmia* only

occurred in two or three cases, and in these the patients were receiving a liberal allowance of wine, arrowroot, and beef tea daily. As a rule, it may be said, that the longer a patient remained in collapse the more liable was he to pulmonary complication during reaction. Probably for this reason: during collapse the blood, directly acted upon by the morbid poison, and still further spoiled by the partial arrest of the normal respiratory changes which this poison induces, becomes surcharged with unoxidized material. Where there has been a more or less complete elimination of the blood poison, when the branches of the pulmonary artery (which we assume to have been in a state of spasm) commence to relax, and the blood is allowed to flow more freely into the pulmonary capillaries, it becomes necessary, if it would go further, that the amount of carbonic acid eliminated should be equal to the amount conveyed to the lungs. Where elimination fails to effect this, the blood, whose onward movement now mainly depends upon the proper performance of these chemical changes, refuses to flow. Congestion occurs. We can easily understand how the tendency to this congestion is increased in direct proportion to the duration of collapse and to the amount of unoxidised material introduced from without. As already remarked, alcohol and opium had previously been freely administered in a large proportion of our cases. In many too, we are willing to confess, there was a too early exhibition of nitrogenous food. This was sometimes ordered by ourselves; but was oftener given by the nurses, who were fearful lest their charges should sink for want of nourishment. We have no hesitation in saying, however, that nitrogenous food invariably did harm when administered during collapse or indeed during reaction, until by the flow of urine, &c., we had evidence that the respiratory changes were being briskly effected. Even supposing that the patient in cholera collapse required food, it is certain that this could not be digested or assimilated; and, supposing none of it found its way into the blood, it must lie as an uneasy load and increase the feeling of oppression by embarrassing those vital

functions which it cannot sustain. Any reduction of temperature during reaction, failure in the force or volume of the pulse, or sinking of the eyeballs we looked upon as signs of approaching congestion. Drowsiness or slight coma preceded nearly every case of congestion. This was not due to uræmia, as water was passed more or less freely in nearly every case, but to the accumulation of carbonic acid in the blood as already stated.

*Remarks upon the remedies employed.*—Castor oil. It will be seen from an examination of our cases that the quantity of oil administered varied greatly. When the natural powers of elimination were pretty active less sufficed, and *vice versâ*. Where vomiting was frequent and energetic the oil was so much the oftener repeated. The amount was also regulated by the stamina of the patient and by the treatment adopted at the commencement of the attack. When there was no vomiting the oil was administered at longer intervals, and assisted by enemata, so as to guard against the possibility of over-purgation. We found acidulated or simple cold water the best vehicle for the administration of the oil. It has been said that in cases where the administration of castor oil was followed by good results, these were possibly due to some alterative influence exerted by it on the intestinal mucous membrane. We never saw any benefit result from its use, except where it excited the intestinal canal to expel its contents. In some cases a pint of oil was administered in twenty-four hours without producing purgation. Surely in these we ought to have had some evidence of the peculiar "castor-oilative" influence of the remedy, if such existed. On the contrary, they all proved fatal, the patients dying with distended bowels. Turpentine we considered a valuable adjunct with castor oil. In cases where the stools were tinged, however slightly, with blood, the oil was omitted, and injections of starch and laudanum given. Almost every case had something peculiar to itself, which modified its treatment. The safest rule in using castor oil is that there should be no general rule at all. The object for which it is given must be borne in

mind. This is not to *increase* the amount of morbid material thrown from the blood into the intestines—"the drain of fluid"—but merely to assist nature in her endeavours to expel this material from the intestines, and so prevent its working further mischief.

*Emetics.*—The value of emetics in the treatment of cholera cannot, we believe, be over-estimated. We found them particularly useful in freeing the stomach of the quantities of opiates and stimulants which had in the majority of our cases been previously administered. The brisk action of a mustard emetic often relieved that feeling of oppression complained of by cholera patients. It may have produced its good effects in these cases by freeing an over-distended stomach, and thus allowing the diaphragm full play, or by throwing out the products of the morbid poison from the stomach. We have seen patients almost asphyxiated improve immediately after the action of an emetic. This may have been owing partly to the mechanical impulse given to the circulation in the series of expiratory efforts accompanying vomiting, and partly to a relaxation of pulmonary arterial spasm probably produced by the *hot water* in which the mustard was suspended. It has been remarked by some of the older writers on cholera, "that collapse was rarely complete till active vomiting had ceased; and it was suggested that such vomiting might tend to obviate the remora of the blood." We made no trial of tartar emetic or any nauseating emetics.<sup>1</sup> *Venesection* was only had recourse to in four cases, and in these it was as a *dernier ressort*. The patients were allowed to remain in collapse too long. Effort after effort was made to restore the eliminative process, under the belief that if this were effected the patient's chances of

<sup>1</sup> It is worthy of remark that in those cases of asphyxia produced by the colliery "choke damp" the usual remedy of the miner is a hot water and mustard emetic.

"The muscular exertion which the frame undergoes in a succession of expiratory actions has the tendency to equalise the distribution and increase the stimulating qualities of the blood."—*Holland's Inquiry into the Laws of Life*.

recovery were greatly increased. Failing in this venesection was resorted to. But the unoxidized and poisoned blood then feebly issuing from the lungs refused to flow. We believe that the abstraction of blood in these cases was the only method by which we could have restored the discharges; as it would, by relieving the pulmonary embarrassment, and freeing the circulation, have increased the excretory power of the intestinal mucous membrane. In similar cases we should resort to blood-letting much earlier.

*Alcoholic stimulants*, except at the commencement of the epidemic were rarely given to a patient in collapse. We have already stated that in many of our cases we consider alcoholic stimulants were much too soon administered during reaction. Ammonia was a very valuable diffusible stimulant where a stimulant was required. *Enemata*.—We found very marked benefit often follow the use of hot injections. At the commencement of the epidemic we used strong saline injections, but we found hot water answer equally well. The temperature of the water was generally about 120° F. It has been said that these hot enemata warm the blood, and thus lessen arterial spasm. This may be so; but we believe that the good effects which follow their use are due as much to the purgation they excite as to any "dilution" or warming of the blood. After free purgation had been induced warm bland injections often relieved the sinking feeling of emptiness in the bowels. *Application of heat*.—We endeavoured to restore warmth by hot bottles, warm flannels, friction with stimulating oils, &c. &c. Warm baths were never tried. In collapse we had often great difficulty in applying heat on account of the jactitations and restlessness of the patient. We think, indeed, that it is open to question whether the forcible confining of the patient in a warm atmosphere at this time was productive of good results or the reverse. *Chloroform* was often administered with the object of relieving the cramps which so generally attend the disease in some stage. Relief appeared to follow its use in many cases, but we believe its free administration tended to prevent pulmonary elimination and increase the collapse.



This, indeed, may have been the very method by which it gave "relief;" because during extreme collapse there is "a torpid condition which seems to be incompatible with cramp or acute pain." *Thermometry*.—The lowest temperature we recorded was 92° F. in the axilla. The thermometer always indicated a lower temperature when placed in the mouth, but this was no measure of the general temperature of the body, or of the degree of collapse. Fluctuations in temperature were so sudden and so frequent that little information was to be derived even from the most carefully recorded indications. We observed, however, the marked decrease of temperature that followed any lengthened arrest in the process of elimination.

*Does the foregoing record of cases give support to the hypothesis which attributes the collapse of cholera to a drain of fluid from the blood?*—It is a necessary and legitimate consequence of this theory that there should be a *direct* relation between the cause and the effect. Almost every writer on cholera has stated that there is *no such* relation; but that if any relation exist it is rather of an *inverse* kind. Accurate observers who have studied the disease in its eastern home (*Scot, Orton, Annesley, Martin, &c.*) speak strongly on this point. Our experience is in strict agreement with that of Dr. Parkes when he says "that, exclusive of the mildest forms of the disease, a case with little vomiting or purging is more malignant, and more rapidly fatal than one in which these discharges are prominent symptoms." We often had the utmost difficulty in *producing purgation* in a collapsed patient, and not a few of our cases proved fatal without there having been either vomiting or purging from the time collapse set in, notwithstanding that very large quantities of strong purgatives had been administered. A patient rarely fell into collapse when the evacuations continued uninterruptedly from the commencement of the attack; but any lengthened arrest in the process of elimination was certain to be followed by algide symptoms more or less intense according to the amount of poison previously eliminated, and to the constitutional susceptibility of the patient.

When a patient who had been freely purged for some hours was brought to hospital in a state of collapse, it seemed very natural to attribute the collapse to the drain produced by the purging, and very easy to build a pathological edifice with this assumption for its foundation; but, by administering purgatives and thus keeping up the drain, the collapse, strange to say, gradually lessened and finally disappeared. Any lengthened cessation from vomiting and purging during collapse we looked upon as a sign of most serious import. An arrest in elimination may arise in two ways. First, it may be caused by the administration of therapeutical agents which have a directly constringing effect upon the intestinal canal—a mode of causation unhappily too frequent. Second, it may be caused by pulmonary obstruction produced by the direct action of the morbid poison in the blood. When the arrest is directly produced by the first method it is rendered greater by the indirect action of the second; because when the evacuations are arrested by the direct action of astringents, the blood already vitiated is still further spoiled by the multiplication and reabsorption of the morbid materials now pent within the bowels; it passes through the lungs in streams still feebler than before, and thus proportionately suppresses all the secretory and excretory functions. How much greater the mischief if to the blood at this time there be added large quantities of hydro-carbonized and nitrogenized material! Yet these were precisely the circumstances under which most of our cases were admitted, and as a consequence we often failed in restoring the excretory action of the intestinal mucous membrane.<sup>1</sup>

*Is there a natural mode of recovery?*—That there is a natural mode of recovery in most diseases, we presume few will deny. Cholera, then, is no exception to this rule. Does recovery from it depend upon an arrest or a continuance of the intestinal evacuations? Upon the answer given to this

<sup>1</sup> [In a previous paper ('Lancet,' Aug. 18th, 1866) it was shown that out of seventeen deaths that occurred from cholera "nine were cases in which neither emesis nor purgation could be produced."—J. W. M'C.]

question will depend, in a great measure, the pathological creed held. There is, we believe, no case on record of a patient having recovered from an attack of cholera without having experienced more or less the peculiar discharges of that disease. Upon looking over our record this will be seen to characterize every case of recovery. Whatever the treatment adopted the result was the same—*recovery never occurred without the continuance of the intestinal discharges or their restoration if previously arrested.*<sup>1</sup> But if the collapse of cholera be produced by the drain of fluid from the blood consequent upon these discharges, surely recovery ought to be preceded by their *arrest*, and not by their continuance. Fatal collapse, on the other hand, invariably followed when the discharges could not be restored. If danger was thus marked “not by the violence of morbid actions, but by the diminution or cessation of natural actions.” Death then may and very often does occur without discharges: recovery never. May we not reasonably look upon the discharges as salutary—as the agents by which nature frees the system of a deadly poison? We believe so, and with this belief we can no longer retain that hypothesis which demands their arrest.

<sup>1</sup> Professor J. Seaton Reid, of Belfast, in a paper upon the cholera outbreak in that town, read before the Ulster Medical Society, October, 1866, states, in speaking of his cases, that he considered they “might be interesting; the more especially as one of them had struggled successfully against the disease, and thus afforded an opportunity of noting particularly the course of his disease.” Upon referring to this case we find that the patient was admitted in a state of collapse; that his bowels did not act for twelve or fourteen hours subsequent to admission, although he had taken ninety grains of calomel, and that he was finally pronounced convalescent *after having vomited about fifty and been purged forty-four times!* After 120 grains of calomel had been administered and very free purgation produced, Prof. Reid considered it advisable to establish “a freer secretion from the intestinal surface by the daily administration of castor oil.” This paper appears in some of the October numbers of the ‘Dublin Medical Press,’ from which it was subsequently reprinted.

A STUDY  
OF THE  
INFLUENCE OF WEATHER AND SEASON  
UPON PUBLIC HEALTH ;

MADE UPON ABOVE 217,000 CASES OF SICKNESS NEWLY  
OCCURRING AT VARIOUS INSTITUTIONS FOR THE  
RELIEF OF THE SICK POOR IN ISLINGTON,  
DURING THE NINE YEARS  
1857—1865.

I. THE INFLUENCE OF ATMOSPHERIC TEMPERATURE.

BY  
EDWARD BALLARD, M.D. LOND., M.R.C.P.,  
MEDICAL OFFICER OF HEALTH FOR ISLINGTON.

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WITH the exception of a paper by Dr. Guy, published in vol. vi of the 'Statistical Journal,' I am not aware of any extensive numerical observations having been made with a view to determine the effect of weather upon public health, except as the latter is measured by the amount of mortality. The indications derivable from the mortality are fallacious, inasmuch as conditions of weather that may bring about a fatal termination of diseases already affecting a population, are not necessarily such as to occasion or favour attacks of illness in persons already in average health. I have frequently been struck, in the preparation of reports upon sickness and mortality, with the great discrepancy in this respect between the tables of sickness and the mortality tables for the same period of time. The inquiry I am about to enter upon, no

doubt, would have been prosecuted long ago, had any national registration of sickness been existent. Several years ago (in 1858) the medical officers of health in the metropolis, feeling how desirable a thing it was that they should be in possession of information as to variations in the amount and character of public sickness, undertook the collection of statistics of sickness, and, returns being made to them from most of the metropolitan districts, the results were published weekly. The aid which they received from the government, however, being withdrawn, this valuable publication ceased. As the medical officer of health in one of its largest parishes, I was a regular contributor to it while it lasted; and when it came to an end, from lack of funds to carry it on, I still continued to collect as before, extending rather than contracting the sources from which I derived information. By the close of 1865 I found myself in possession of a record of over 217,000 cases of sickness, certain acute and readily determined diseases being specially enumerated. In respect of all, I am in possession of a record of the month in which they occurred, and, as respects the greater number, of the week. They were all newly occurring cases of sickness seeking aid either at the hands of parochial surgeons, or at one or other of the dispensaries in Islington; and I have been permitted to include, by the kindness of the surgeon of the establishment, Mr. Bradley, the new cases of sickness happening in the Pentonville convict prison.

The materials at hand are such as to enable me to discuss, with some prospect of success, a considerable number of very important questions; but the first in importance has appeared to me to be the relation which existed between the amount of sickness prevalent at any time and the weather with which it was associated. In doing this I have availed myself of the meteorological tables published by Mr. Glaisher, in the returns of the Registrar General. Their public character, the readiness with which they may be consulted, and their great accuracy, far beyond that of any local tables I could have access to, led me to prefer their use.

Two methods of prosecuting the inquiry were open to me.

One of these was to compare the actual amount of sickness week by week, month by month, quarter by quarter, or year by year, with the meteorological conditions of each of these periods of time; the other, to note the fluctuations only of the sickness observed, and to compare them with the variations which occurred from time to time in atmospherical conditions. Both methods give valuable results and one may be used as confirmatory of the results of the other. But, of the two, the former is open to more fallacy than the latter, inasmuch as the absolute amount of sickness is dependent very much upon long antecedent conditions; so that very different amounts of sickness come to be recorded in seasons which in themselves differ very little in the character of their weather. The latter method is, to some extent it is true, liable to the same objection; but the influence of antecedent seasons is lost sight of where an extensive subdivision of time is had recourse to, and where such short periods with merely their fluctuations of sickness, as taken from very different years and seasons, are mixed together in the same table; note being taken, not of the actual numerical amount of the several meteorological conditions (such as the actual temperature, actual degree of humidity, &c.), but merely of their fluctuations from one short period of time to another. This latter method, then, I propose first of all to adopt, reserving to myself the use of the former where it appears likely to afford valuable information.

Of all the meteorological conditions ordinarily observed, none is more influential than atmospheric temperature; none more constant in its operation, nor more consistent in its mode of operation. I propose then to commence with this element of weather, and first of all to show how it operated in the course of 389 weeks, comparing the variation of mean temperature, week by week, with the fluctuations in general sickness which were associated with it. The materials for this comparison will be furnished by the records of sickness as supplied to me, week by week, in the years 1858 to 1865 from the following sources: viz. the Islington workhouse and infant poor house; the case-books of eight district parochial

surgeons: the books of the Central Holloway Dispensary and of the Islington Dispensary. During these eight years 158,721 new cases of sickness of all kinds came under observation. For a reason to be presently assigned, a few weeks at the close of each year have been excluded from the inquiry, as fallacious in their indications.

Table I represents the fluctuation in general sickness which took place in the course of 196 weeks of rise of mean temperature during the eight years 1858-1865.

Certain weeks immediately about Christmas in each year are excluded from the summary, inasmuch as the account of sickness in my record for those weeks is not to be relied upon as a fair representation of what actually occurred. At and about Christmas I have invariably found that the applications for medical relief at the institutions which supply the basis of my record were greatly and apparently capriciously reduced.

The normal tendency of rises of mean temperature is to occasion increase of sickness in the weeks in which they occur.

In the course of the 196 weeks during which rises of mean temperature of various extent took place, increases of sickness occurred to the total amount of 4246 cases, and decreases to the total amount of 2000 cases.

Thus, for every 1000 cases of increase of sickness there occurred only 471 cases of decrease.

For the purposes of the analyses I have found it convenient arbitrarily to divide both rises and falls of temperature into those which are "slight," or under 2 degrees in extent; "moderate," or to the extent of 2 and less than 5 degrees; and "considerable," to the extent of 5 degrees and upwards.

That this difference as to increase and decrease of sickness was not accidental, but more or less directly the result of the rises of temperature, appears from observing that the increases were greater in proportion to the decreases of sickness according as the extent of the associated rises was greater. Thus—

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*Tables showing the relation between fluctuations of mean weekly temperature and weekly fluctuations of general sickness.*

TABLE I.—Rises.

	Increase of sickness.										Decrease of sickness.					Neither increase nor decrease of sickness.			
	Number of weeks.	Sum of rises.	Mean weekly rise.	Number of weeks.	Sum of rises.	Mean weekly rise.	Frequency of increase per cent. of weeks.	Amount of increased sickness.		Number of weeks.	Sum of rises.	Mean weekly rise.	Frequency of decrease per cent. of weeks.	Amount of decreased sickness.		Number of weeks.	Sum of rises.	Mean weekly rise.	Frequency per cent. of weeks.
								Sum.	Mean.					Sum.	Mean.				
Rises generally.....	196	733.5°	3.74°	121	497.5°	4.11°	61.73	4246	35.09	74	232.5°	3.14°	37.75	2000	27.0	1	3.5°	3.5°	0.52
Slight rises (under 2°)	70	70.7°	1.01°	40	39.0°	0.97°	57.14	1166	29.1	30	31.7°	1.05°	42.86	984	32.8				
Moderate rises (2° and under 5°).....	73	245.0°	3.35°	40	137.8°	3.44°	54.79	1295	32.3	32	103.7°	3.24°	43.84	761	23.7	1	3.5°	3.5°	1.3
Considerable rises (5° and upwards) .....	53	417.8°	7.88°	41	320.7°	7.82°	77.36	1785	43.5	12	97.1°	8.09°	22.64	255	21.2				



TABLE II.—*Falls.*

	Increase of sickness.						Decrease of sickness.						Neither increase nor decrease of sickness.			
	Number of weeks.	Sum of falls.	Mean weekly fall.	Number of weeks.	Sum of falls.	Mean weekly fall.	Frequency of increase per cent. of weeks.	Amount of increased sickness.		Frequency of decrease per cent. of weeks.	Amount of decreased sickness.		Number of weeks.	Sum of falls.	Mean weekly fall.	Frequency per cent. of weeks.
								Sum.	Mean.		Sum.	Mean.				
Falls generally .....	193	700.1°	3.63°	76	260.7°	3.43°	39.38	2253	29.6	58.55	4538	40.2	4	3.1°	0.7°	2.07
Slight falls (under 2°)	58	60.1°	1.03°	26	27.9°	1.07°	44.83	1028	39.5	50.00	960	33.1	3	1.1°	0.3°	5.17
Moderate falls (2° and under 5°).....	87	290.0°	3.33°	34	117.6°	3.45°	39.08	855	25.1	59.77	1823	35.1	1	2.0°	2.0°	1.15
Considerable falls (5° and upwards) .....	48	350.0°	7.29°	16	115.2°	7.20°	33.33	370	23.1	66.67	1755	54.8				

In the course of the 70 weeks of slight rise of temperature, for every 1000 cases of increase of sickness there happened 843 cases of decrease.

In the course of the 73 weeks of moderate rise of temperature, for every 1000 cases of increase of sickness there happened 587 cases of decrease.

In the course of the 53 weeks of considerable rise of temperature, for every 1000 cases of increase of sickness there happened 142 cases of decrease.

Table II represents the fluctuation in general sickness which took place in the course of 193 weeks of fall of mean temperature during the same eight years.

The normal tendency of falls of mean weekly temperature is to occasion decrease of sickness in the weeks in which they occur.

In the course of the 193 weeks during which falls of mean temperature of various extent took place, decreases of sickness occurred to the total amount of 4538 cases, and increases to the total amount of 2253 cases.

Thus for every 1000 cases of decrease of sickness there occurred only 496 cases of increase.

That this difference again was not accidental, but more or less directly the result of the falls of temperature, appears from observing that the decreases were greater in proportion to the increases of sickness according as the extent of the associated falls was greater. Thus,—

In the course of the 58 weeks of slight fall of temperature, for every 1000 cases of decrease of sickness there happened 1070 cases of increase.

In the course of the 87 weeks of moderate fall of temperature, for every 1000 cases of decrease of sickness there happened 469 cases of increase.

In the course of the 48 weeks of considerable fall of temperature, for every 1000 cases of decrease of sickness there happened 210 cases of increase.

It is remarkable, and a point which will be again referred to, that the decreases of sickness in the weeks of slight fall

were altogether less than the increases which took place in these weeks.

Assuming that normally rises and falls of temperature to a similar extent act with a like force in producing fluctuations of sickness, although in opposite directions, we may estimate their normal force, under average conditions in regard to supplementary meteorological states of the atmosphere, so far as our tables are concerned, as follows :

We have upon our tables altogether 389 weeks in which rises of mean temperature took place to the total extent of 733·5 degrees, and in which falls took place to the total extent of 700·1 degrees, making together fluctuations, either by way of rise or fall of 1433·6 degrees.

During the 169 weeks of rise of temperature increases of sickness occurred to the amount of 4246 cases and decreases to the amount of 2000 cases, giving thus a clear gain by way of increase of sickness amounting to 2246 cases.

Again, during the 193 weeks of fall of temperature decreases of sickness occurred to the amount of 4538 cases, and increases to the amount of 2253 cases, giving thus a clear gain by way of decrease of sickness, amounting to 2285 cases.

Adding these normal gains together, we obtain as the total result of the 389 fluctuations of mean temperature corresponding fluctuations of sickness, the clear gain from which by way of increase or decrease amounts to 4531 cases. This is the final result of 1433 degrees of fluctuation of temperature, so that the mean result, under average conditions, of a change of temperature either by way of rise or fall, to the extent of one degree, may be taken to be an increase or a decrease of sickness to the amount of 3·16 cases ; all above or below which must be attributed to the operation of supplementary meteorological conditions co-operating with or antagonising the rises or falls of temperature. Such conditions may, as we shall see, be concurrent with or antecedent to the changes of temperature referred to.

We may now apply this standard of force to the results of the rises and falls as stated upon the tables. Thus,—

There were 196 rises, the sum of which was 733·5 degrees. The clear gain by way of increase of sickness which might have been anticipated under average supplementary conditions is therefore 2317 cases. But, deducting the decreases from the increases of sickness, we obtain the number 2246 cases of increase only, so that the gain of increase of sickness does not reach the estimated amount by 71 cases. I infer from this that in the course of these 196 weeks of rise of temperature in which various supplementary conditions, promotive some of increase and others of decrease of sickness, were operative, an excess of force was exercised by the antagonistic conditions (those promotive of decrease) which may be represented by saying that it was equal to what would, under average conditions, be exerted by 22·47 degrees of fall of mean temperature or by a mean weekly reduction of temperature to the extent of 0·11 degrees.

Applying the same standard to the results of slight, moderate, and considerable rises severally, we find—

1st. *As to slight rises.*—There were 70 such rises, the sum of which was 70·7 degrees, the clear gain by way of increase from which would, under average conditions, be 223 cases. But, deducting the decreases which occurred from the cases of increase, we obtain as the actual clear gain only 182 cases, so that the gain does not reach the estimated amount by 41 cases. Thus, as respects these 70 weeks of slight rise, we find that in their course an excess of force was exercised on the part of the supplementary antagonistic conditions, which may be represented by saying that it was equal to what, under average conditions, would be exerted by 12·97 degrees of fall of mean temperature, or by a mean weekly reduction of temperature to the extent of 0·18 degrees.

2nd. *As to moderate rises.*—There were 73 such rises, the sum of which was 245·0 degrees, the clear gain by way of increase of sickness from which would, under average conditions, be 774 cases. But, deducting the decreases which occurred from the cases of increase, we obtain as the actual clear gain only 534 cases, so that the gain does not reach the

estimated amount by 240 cases. Hence it is to be inferred as respects these 73 weeks of moderate rise that in their course an excess of force was exercised on the part of the supplementary antagonistic conditions equal to what under average conditions would be exerted by 75·95 degrees of fall of mean temperature, or a mean weekly reduction of temperature to the extent of 1·04 degrees.

3rd. *As to considerable rises.*—There were 53 such rises, the sum of which was 417·8 degrees, the clear gain by way of increase of sickness from which would, under average circumstances, be 1320 cases. But, deducting the cases of decrease of sickness from those of increase, we obtain as actual clear gain 1530 cases, so that the gain exceeds the estimated amount by 210 cases. Hence it may be inferred, as respects these 53 weeks of considerable rise, that in their course an excess of force was exercised on the part of the supplementary co-operative conditions (promotive of increase of sickness) equal to what, under average conditions, would be exerted by 66·46 degrees of rise of mean temperature, or by a further mean weekly rise of 1·25 degrees.

Again. There were 193 falls, the sum of which was 700·1 degrees, the clear gain by way of decrease of sickness from which would, under average circumstances, be 2212 cases. But, now deducting the cases of increase of sickness on the table from those of decrease, we obtain an actual clear gain of 2285 cases, so that the gain exceeds the estimated amount by 73 cases. Hence it may be inferred that in the course of these 193 weeks of fall of temperature, an excess of force was exercised on the part of the supplementary co-operative conditions (promotive of decrease of sickness) equal to what under average circumstances would be exerted by 23 degrees of fall of mean temperature, or by a further mean weekly fall to the extent of 0·12 degrees. Then—

1st. *As to slight falls.*—There were 58 such falls, the sum of which was 60·1 degrees, the clear gain by way of decrease of sickness from which would, under average circumstances, be 190 cases. But we observe on consulting Table 2 that so far from there being any such gain the cases of decrease of

sickness were less numerous than those of increase by 68 cases. Hence we must infer that in the course of these 58 weeks of slight fall an excess of force was exercised on the part of the supplementary antagonistic conditions (promotive of increase of sickness) sufficient not only to neutralize the normal decrease of 190 cases, *but to produce 68 cases of increase also*—a force thus capable of increasing sickness virtually by 258 cases, and therefore equal to what under average circumstances would be exerted by 81·65 degrees of rise of mean temperature, or by a mean weekly rise to the extent of 1·40 degrees.

2nd. *As to moderate falls.*—There were 87 such falls, the sum of which was 290 degrees, the clear gain by way of decrease of sickness from which would, under average circumstances, be 916 cases. But, deducting the cases of increase from those of decrease we obtain as actual clear gain a decrease of 968 cases, so that the gain exceeds the estimated amount by 52 cases. Hence it may be inferred, as respects these 87 weeks of moderate fall of temperature, that, in their course, an excess of force was exercised by the supplementary co-operative conditions (promotive of decrease) equal to what under average circumstances would be exerted by 16·45 degrees of fall of mean temperature, or by a further mean weekly fall to the extent of 0·18 degrees.

3rd. *As to considerable falls.*—There were 48 such falls, the sum of which was 350 degrees, the clear gain by way of decrease of sickness from which would, under average circumstances, be 1106 cases. But, deducting the cases of increase of sickness from those of decrease, we obtain as actual clear gain a decrease of 1385 cases, so that the gain exceeds the estimated amount by 279 cases. Hence it may be inferred as respects these 48 weeks of considerable fall that, in their course, an excess of force was exercised by the supplementary co-operative conditions equal to what, under average circumstances, would be exerted by 88·29 degrees of fall of mean temperature, or by a further mean weekly fall to the extent of 1·84 degrees.

A deduction, which there will be occasion to use presently, may here be drawn, viz., that in the weeks of rise generally, and in the weeks of fall generally, the supplementary causes of increase and decrease of sickness are balanced in the same manner. In both the supplementary causes of decrease of sickness operate in excess of those promotive of increase of sickness, and as nearly as possible with the same force.

**RELATION OF RISES AND FALLS OF MEAN WEEKLY TEMPERATURE TO THE FREQUENCY OF OCCURRENCE OF INCREASE AND DECREASE OF SICKNESS.**

**1. *Rises.***

1st. Rises of mean weekly temperature were associated with increase of sickness much more frequently than with decrease of sickness.

Increase of sickness occurred in 61·73 per cent. of the weeks in which a rise of temperature took place; decrease in 37·75 per cent.

2nd. That the rises themselves were influential in producing the greater frequency with which increase of sickness took place is shown by the observation that the frequency with which increase of sickness occurred was greater when the extent of the rises was great than when it was comparatively small.

The frequency of increase of sickness in the 53 weeks of considerable rise of mean temperature (mean 7·88 degrees) was 77·36 per cent.; in the 143 weeks of slight and moderate rise of mean temperature (mean 2·2 degrees), 55·92 per cent. There was one week in which neither increase nor decrease of sickness occurred.

**2. *Falls.***

1st. Falls of mean weekly temperature were associated with decrease of sickness much more frequently than with increase of sickness.

Decrease of sickness occurred in 58·54 per cent. of the

weeks in which a fall of temperature took place ; increase of sickness in 39·37 per cent.

2nd. That the falls themselves were influential in producing the greater frequency with which decrease of sickness took place is shown by the observation that the frequency with which decrease of sickness occurred was greater when the extent of the falls was great than when it was comparatively small.

The frequency of decrease of sickness in the 48 weeks of considerable fall of mean temperature (mean 7·29 degrees) was 66·66 per cent. ; in the 87 weeks of moderate fall of mean temperature (mean 3·33 degrees), 59·77 per cent. ; in the 58 weeks of slight fall of mean temperature (mean 1·03 degrees), 50 per cent.

One striking fact at least shows that something else besides the change of temperature influenced the occurrence of increase or decrease of sickness in the weeks under examination, viz., that in the weeks of moderate rise, the frequency with which increase of sickness occurred was less than in the weeks of slight rise. If it were otherwise increase of sickness would always be associated with rises of temperature, and decrease with falls. That decrease of sickness ever occurs with a rise of temperature shows that sometimes supplementary causes are in operation capable of overpowering the influence of the rises ; and that increase of sickness ever occurs with falls of temperature shows that supplementary causes are sometimes in operation capable of overpowering the influence of the falls. In any selected week, beside the rise or fall of temperature, supplementary causes both of increase and of decrease of sickness must be believed to be existent, and according as those promotive of increase or those promotive of decrease are in excess or predominant, there will be a tendency either to increase of sickness or to decrease. In the one case the supplementary causes in excess will tend to co-operate with rises and assist them in increasing sickness, or else tend to antagonise the falls and prevent them from decreasing sickness ; and in the other case the supplementary causes in



excess will tend to co-operate with falls and assist them in decreasing sickness, or else tend to antagonise rises and prevent them from increasing sickness. Whether in any week sickness will be increased or decreased then will depend upon the manner in which all these influences are balanced as regards their force. The factors are the extent of the rise or fall of temperature (their force); the predominance of supplementary conditions tending to increase or decrease sickness; and the degree (force) of their excess or predominance.

We will now, therefore, endeavour to discover as respects the weeks of rise or fall of mean temperature how far the occurrence of increase of sickness with rises, and of decrease of sickness with falls, as recorded upon our tables, was dependent upon the rises or falls themselves, and how far upon the predominance, in each set of weeks, of supplementary causes of increase or decrease of sickness; and again how far these latter causes operated in producing decrease of sickness with rises of temperature, and increase with falls.

Certain assumptions will have to be made, the probability of which will govern the trust to be placed in the results of the inquiry.

The first is that, just as rises and falls of temperature are about equal in frequency in the course of 389 weeks, and as the distribution of force of the various supplementary causes of increase and decrease is altogether similar in the 196 weeks of rise and the 193 weeks of fall, so also may the frequency with which the supplementary causes of increase and decrease are severally in excess in the weeks of rise or fall of temperature be regarded as similar.

The second is that this assumption may be accepted as applying to rises and falls of all degrees of extent; that is to say, whatever may be determined upon as the frequency with which supplementary causes of increase and decrease of sickness are in excess in weeks of rise and fall of temperature generally, that shall be regarded as the frequency with which either predominates in the weeks of slight, moderate, or considerable rise or fall of temperature respectively.

Let us endeavour, then, to ascertain with what relative frequency the supplementary causes of increase and decrease of sickness predominated in the 389 weeks.

First, looking at Table 1, we see that there were 38·27 per cent. of weeks of rise, in which the supplementary causes of decrease of sickness predominated over all causes of increase of sickness, including rises of temperature.

Secondly, looking at Table 2, we see that there were 41·45 per cent. of weeks of fall, in which the supplementary causes of increase of sickness predominated over all causes of decrease of sickness, including falls of temperature.

There remain 20·28 per cent. of weeks in which it is probable that a similar predominance of one class of causes over the other existed, although not sufficient to neutralize the obvious operation of the rises and falls of temperature. Distributing the frequency of predominance of supplementary causes similarly in these weeks we have 9·74 per cent. additional of weeks in which causes of decrease predominated, and 10·54 per cent. in which causes of increase of sickness predominated.

Hence we may regard it as probable that in the 389 weeks, whether of rise or fall of temperature, supplementary causes of increase of sickness predominated in operation in 52 per cent., and of decrease in 48 per cent.

But we have seen that both in weeks of rise and fall of temperature, the sum of force of operation of the supplementary causes of decrease of sickness was greater than that of the supplementary causes of increase.

Hence a further deduction that the supplementary causes of increase of sickness operate more frequently but with less force than the supplementary causes of decrease, while the supplementary causes of decrease of sickness act with more force but less frequency than the supplementary causes of increase.

#### 1. *As to rises of mean temperature.*

Had there been no rises of mean temperature in the course

Applying the same method to weeks of rise of temperature of different degrees of extent we may tabulate the results also as follows :—

*Weeks of slight rise.*

Weeks.	Proportion of predominance of supplementary causes of increase or decrease.
57.14 per cent. increase.	48.00 {
42.86 per cent. decrease.	
	<p>In 52.00 per cent., increase of sickness occurred from joint operation of slight rises and other supplementary causes of increase.</p> <p>In 5.14 per cent., increase of sickness occurred from operation of slight rises alone overcoming the obstacle afforded by predominance of supplementary causes of decrease.</p> <p>In 42.86 per cent., decrease of sickness occurred from predominant operation of supplementary cause of decrease, overcoming the influence of the rises and producing manifest decrease of sickness.</p>
100.00	100.00

*Weeks of moderate rise.*

Weeks.	Proportion of predominance of supplementary causes of increase or decrease.
54.79 per cent. increase.	48.00 {
45.21 per cent. decrease or no increase.	
	<p>In 52.00 per cent., increase of sickness occurred from joint operations of moderate rises and other supplementary causes of increase.</p> <p>In 2.79 per cent., increase of sickness occurred from operation of moderate rises alone overcoming the obstacle afforded by predominance of supplementary causes of decrease.</p> <p>In 43.84 per cent., decrease of sickness occurred from predominant operations of supplementary causes of decrease overcoming the influence of the rises and producing manifest decrease of sickness.</p> <p>In 1.37 per cent., merely no increase of sickness occurred from same cause, but not forcible enough to do more than neutralize the rises.</p>
100.00	100.00

of the 196 weeks we should still, upon our estimate of the frequency of predominance of the supplementary causes of increase of sickness, have met with increase of sickness in 52 per cent. of those weeks.

But the actual frequency of increase was 61·73 per cent., that is, there was an excess of frequency of increase in these weeks of rise to the extent of 9·73 per cent. This excess of frequency may be held to be due therefore to the superior influence of the rises of temperature overcoming all antagonistic influences opposed to them; while in 52 per cent. of the weeks of rise the rises merely co-operated with other predominating causes of increase, the increase of sickness that occurred being the joint result of the rises of temperature and these predominating supplementary influences.

Then we have 38·27 per cent. of weeks in which the causes of decrease predominated in force to such an extent as to neutralize the operation of the rises of temperature. In 37·75 of these weeks the force of these causes was sufficient not only to do this but also to occasion more or less manifest decrease of sickness.

Placing this analysis of force in a tabular form we may state it thus:—

Weeks.	Proportion of predominance of supplementary causes of increase or decrease.
61·73 per cent. increase of sickness.	In 52·00 per cent. of weeks, increase of sickness occurred from joint operation of rises and other supplementary causes of increase.
	In 9·73 per cent., increase of sickness occurred from operation of rises alone overcoming the obstacle afforded by predominance of supplementary cause of decrease.
38·27 per cent. decrease of sickness, or no increase.	In 37·75 per cent., decrease of sickness occurred from predominant operation of supplementary causes of decrease overcoming the influence of the rises, and producing manifest decrease.
	In 0·52 per cent., merely no increase of sickness occurred from same cause, but not forcible enough to do more than neutralize the rise.
100·00	100·00

Applying the same method to weeks of rise of temperature of different degrees of extent we may tabulate the results also as follows :—

*Weeks of slight rise.*

Weeks.	Proportion of predominance of supplementary causes of increase or decrease.
57·14 per cent. increase.	In 52·00 per cent., increase of sickness occurred from joint operation of slight rises and other supplementary causes of increase. In 5·14 per cent., increase of sickness occurred from operation of slight rises alone overcoming the obstacle afforded by predominance of supplementary causes of decrease. 48·00 In 42·86 per cent., decrease of sickness occurred from predominant operation of supplementary cause of decrease, overcoming the influence of the rises and producing manifest decrease of sickness.
42·86 per cent. decrease.	
<hr/> 100 00	<hr/> 100·00

*Weeks of moderate rise.*

Weeks.	Proportion of predominance of supplementary causes of increase or decrease.
54·79 per cent. increase.	In 52·00 per cent., increase of sickness occurred from joint operations of moderate rises and other supplementary causes of increase. In 2·79 per cent., increase of sickness occurred from operation of moderate rises alone overcoming the obstacle afforded by predominance of supplementary causes of decrease. 48·00 In 43·84 per cent., decrease of sickness occurred from predominant operations of supplementary causes of decrease overcoming the influence of the rises and producing manifest decrease of sickness. In 1·37 per cent., merely no increase of sickness occurred from same cause, but not forcible enough to do more than neutralize the rises.
45·21 per cent. decrease or no increase.	
<hr/> 100·00	<hr/> 100·00

of the 196 weeks we should still, upon our estimate of the frequency of predominance of the supplementary causes of increase of sickness, have met with increase of sickness in 52 per cent. of those weeks.

But the actual frequency of increase was 61·73 per cent., that is, there was an excess of frequency of increase in these weeks of rise to the extent of 9·73 per cent. This excess of frequency may be held to be due therefore to the superior influence of the rises of temperature overcoming all antagonistic influences opposed to them; while in 52 per cent. of the weeks of rise the rises merely co-operated with other predominating causes of increase, the increase of sickness that occurred being the joint result of the rises of temperature and these predominating supplementary influences.

Then we have 38·27 per cent. of weeks in which the causes of decrease predominated in force to such an extent as to neutralize the operation of the rises of temperature. In 37·75 of these weeks the force of these causes was sufficient not only to do this but also to occasion more or less manifest decrease of sickness.

Placing this analysis of force in a tabular form we may state it thus:—

Weeks.	Proportion of predominance of supplementary causes of increase or decrease.
61·73 per cent. increase of sickness.	In 52·00 per cent. of weeks, increase of sickness occurred from joint operation of rises and other supplementary causes of increase.
	In 9·73 per cent., increase of sickness occurred from operation of rises alone overcoming the obstacle afforded by predominance of supplementary cause of decrease.
38·27 per cent. decrease of sickness, or no increase.	In 37·75 per cent., decrease of sickness occurred from predominant operation of supplementary causes of decrease overcoming the influence of the rises, and producing manifest decrease.
	In 0·52 per cent., merely no increase of sickness occurred from same cause, but not forcible enough to do more than neutralize the rise.
<hr/> 100·00	<hr/> 100·00

Applying the same method to weeks of rise of temperature of different degrees of extent we may tabulate the results also as follows :—

*Weeks of slight rise.*

Weeks.	Proportion of predominance of supplementary causes of increase or decrease.
57.14 per cent. increase.	{ In 52.00 per cent., increase of sickness occurred from joint operation of slight rises and other supplementary causes of increase. { In 5.14 per cent., increase of sickness occurred from operation of slight rises alone overcoming the obstacle afforded by predominance of supplementary causes of decrease. 48.00 { In 42.86 per cent., decrease of sickness occurred from predominant operation of supplementary cause of decrease, overcoming the influence of the rises and producing manifest decrease of sickness.
42.86 per cent. decrease.	
<hr/> 100.00	<hr/> 100.00

*Weeks of moderate rise.*

Weeks.	Proportion of predominance of supplementary causes of increase or decrease.
54.79 per cent. increase.	{ In 52.00 per cent., increase of sickness occurred from joint operations of moderate rises and other supplementary causes of increase. { In 2.79 per cent., increase of sickness occurred from operation of moderate rises alone overcoming the obstacle afforded by predominance of supplementary causes of decrease. 48.00 { In 43.84 per cent., decrease of sickness occurred from predominant operations of supplementary causes of decrease overcoming the influence of the rises and producing manifest decrease of sickness. { In 1.37 per cent., merely no increase of sickness occurred from same cause, but not forcible enough to do more than neutralize the rises.
45.21 per cent. decrease or no increase.	
<hr/> 100.00	<hr/> 100.00

*Weeks of considerable rise.*

Weeks.	Proportion of predominance of supplementary causes of increase or decrease.
77.36 per cent. increase.	In 52.00 per cent., increase of sickness occurred from joint operation of considerable rises and other supplementary causes of increase. In 25.36 per cent., increase of sickness occurred from operation of considerable rises alone overcoming the obstacles afforded by predominance of supplementary causes of decrease. In 22.64 per cent., decrease of sickness occurred from predominant operation of supplementary causes of decrease overcoming the influence of the considerable rises, and producing manifest decrease of sickness.
22.64 per cent. decrease.	
100.00	100.00

*2. As to falls of mean temperature.*

Had there been no falls of mean temperature in the course of the 193 weeks we should, upon our estimate of the frequency of predominance of the supplementary causes of decrease of sickness, have met with decrease of sickness in 48 per cent. of those weeks.

But the actual frequency of decrease was 58.55 per cent., that is, there was an excess of frequency of decrease in these weeks of fall to the extent of 10.55 per cent. This excess of frequency may be held to be due, therefore, to the superior influence of the falls of temperature, overcoming all antagonistic influences opposed to them; while in 48 per cent. of the weeks of fall, the falls merely co-operated with other predominating causes of decrease, the decrease of sickness that occurred being the joint result of the falls of temperature and these predominating supplementary influences.

Thus we have 41.45 per cent. of weeks in which the causes of increase of sickness predominated in force to such an extent as to neutralize the operation of the falls of temperature. In 39.38 of these weeks the force of these causes was sufficient not only to do this, but also to occasion more or less manifest increase of sickness.



Tabulating this statement we may place it thus :—

Weeks.	Proportion of predominance of supplementary causes of increase or decrease.	
58.55 per cent. decrease of sickness.	52.00	In 48.00 per cent. of the weeks of fall, decrease of sickness occurred from the joint operation of falls and other supplementary causes of decrease.
41.45 per cent. increase of sickness or no decrease.		In 10.55 per cent., decrease of sickness occurred from operation of falls alone overcoming the obstacles afforded by the predominance of supplementary causes of increase.
		In 39.38 per cent., increase of sickness occurred from predominant operation of supplementary causes of increase overcoming the influence of the falls and producing manifest increase of sickness.
		In 2.07 per cent., no decrease of sickness merely took place from same cause, but not forcible enough to do more than neutralize the falls.
100.00	100.00	

Applying the same method to weeks of fall of temperature of different degrees of extent we may tabulate the results as follows :

*Weeks of slight fall.*

Weeks.	Proportion of predominance of supplementary causes of increase or decrease.	
50.00 per cent. decrease of sickness.	52.00	In 48.00 per cent., increase of sickness occurred from joint operation of slight falls and other supplementary causes of decrease.
50.00 per cent. increase of sickness or no decrease.		In 2.00 per cent., decrease of sickness occurred from operation of slight falls alone overcoming the obstacle afforded by the predominance of supplementary causes of increase of sickness.
		In 44.83 per cent., increase of sickness occurred from predominant operation of supplementary causes of increase overcoming the influence of the falls and producing manifest increase.
		In 5.17 per cent., no decrease merely took place from same cause, but not forcible enough to do more than neutralize the falls.
100.00	100.00	

*Weeks of moderate fall.*

Weeks.	Proportion of predominance of supplementary causes of increase or decrease.
59-77 per cent. decrease of sickness.	In 48-00 per cent., decrease of sickness occurred from joint operation of moderate falls and other supplementary causes of decrease. In 11-77 per cent., decrease of sickness occurred from operation of moderate falls alone overcoming the obstacle afforded by the predominance of supplementary causes of increase of sickness. In 39-08 per cent., increase of sickness occurred from predominant operation of supplementary causes of increase overcoming the influence of the falls and producing manifest increase. In 1-15 per cent., no decrease of sickness merely took place from same cause, but not forcible enough to do more than neutralize the falls.
40-23 per cent. increase or no decrease of sickness.	
100-00	100-00

*Weeks of considerable fall.*

Weeks.	Proportion of predominance of supplementary causes of increase or decrease.
66-67 per cent. decrease of sickness.	In 48-00 per cent., decrease of sickness occurred from joint operation of considerable falls and other supplementary causes of decrease. In 18-67 per cent., decrease of sickness occurred from operation of considerable falls alone overcoming the obstacle afforded by the predominance of supplementary causes of increase of sickness. In 33-33 per cent., increase of sickness occurred from predominant operation of supplementary causes of increase, overcoming the influence of the falls and producing manifest increase.
33-33 per cent. increase of sickness.	
100-00	100-00

RELATION OF RISES AND FALLS OF MEAN WEEKLY TEMPERATURE TO THE AMOUNT OF INCREASE AND DECREASE OF SICKNESS WHEN EITHER TOOK PLACE.

*1. Rises.*

The mean amount of increase of sickness in the weeks of rise of temperature in which increase took place was greater

than that of decrease in the weeks in which decrease of sickness took place.

Thus the total amount of increase of sickness in the 121 weeks in which it occurred was 4246 cases—mean, 35·09 cases per week; and the total amount of decrease of sickness in the 74 weeks in which it occurred was 2,000 cases—mean, 27 cases per week.

The influence of the rise of temperature in producing the increase of sickness that was observed, and in checking the operation of the causes of decrease, is shown by the fact that the mean weekly amount of all the weekly increases of sickness that occurred in the course of the 389 weeks was 32·99 cases, and of all the decreases 34·96 cases.

In the case of the slight rises, however, it is observable that the mean amount of weekly decrease of sickness when it occurred was greater than that of increase of sickness when increase took place.

*A. As to the amount of increase of sickness.*

1st. The amount of increase of sickness when it occurred was very much governed by the extent of the rises of mean temperature with which it was associated.

This appears from observing that the mean weekly amount of increase of sickness was greater in those weeks in which the extent of the rise of temperature was great than those in which it was comparatively small. Thus, in the 40 weeks of slight rise in which increase of sickness occurred, the mean amount was 29·1 cases; in the 40 weeks of moderate rise, 32·3 cases; in the 41 weeks of considerable rise, 43·5 cases.

2nd. The amount of increase of sickness, when it occurred, was also to a certain degree dependent upon other supplementary meteorological conditions.

The mean extent of the 121 rises of mean weekly temperature with which increase of sickness was associated was 4·11 degrees—the sum of all the rises together being 497·5 degrees. Now, assuming, as we have previously, that a rise of mean temperature to the extent of one degree is in our table equivalent to an increase of sickness to the amount of

3·16 cases, the amount of increase of sickness which under average conditions would have occurred in these 121 weeks is only 1572 cases. But the actual increase that occurred was 4246 cases. Hence in these 121 weeks an amount of increase of sickness to the extent of 2674 cases took place which the extent of the rises of temperature will not account for, and which must therefore be regarded as due to the operation of those predominant supplementary causes of increase of sickness which we have already referred to as co-operating with the rises in most of these weeks. Of the 35·09 cases of mean weekly increase of sickness then only 12·99 can be held due to the operation of the rises, while the larger proportion of 22·10 cases must be otherwise accounted for. The combined force of operation of the supplementary causes of increase in these weeks was then equal to what, under average circumstances, would be exerted by 846·20 degrees of rise of mean temperature, or by a further mean weekly rise of 6·99 degrees.

*As to slight rises.*—The mean extent of the 40 slight rises of temperature with which increase of sickness was associated was 0·97 degrees, the sum of all these rises together being 39 degrees. Under average conditions the amount of increase of sickness which would have occurred in these 40 weeks is 123 cases only. But the actual increase was 1166 cases, leaving thus 1043 cases due to the predominant operation of supplementary causes of increase co-operating in the majority of weeks with the rises. Of the 29·1 mean weekly increase in these weeks then only 3·1 cases can be regarded as due to the rises of temperature, while 26 cases must be attributed to the operation of other predominant causes of increase of sickness. The combined force of operation of these supplementary causes was then equal to what, under average circumstances, would be exerted by 330·06 degrees of rise of mean temperature, or by a further mean weekly rise of 8·25 degrees.

*As to moderate rises.*—The mean extent of the 40 moderate rises of temperature with which increase of sickness was associated was 3·44 degrees, the sum of all these rises together

being 137·8 degrees. Under average conditions, the amount of increase of sickness which would have occurred in these 40 weeks is 435 cases. But the actual increase which occurred was 1,295 cases, leaving thus 860 cases due to the predominant operation of supplementary causes of increase of sickness. Of the 32·3 mean cases of weekly increase in these weeks then only 10·9 cases were due to the rise of temperature, leaving 21·4 cases attributable to the operation of the predominant supplementary causes of increase. The combined force of operation of these supplementary causes was then equal to what, under average circumstances, would be exerted by 272·15 degrees of rise of temperature, or by a further mean weekly rise of 6·80 degrees.

*As to considerable rises.*—The mean extent of the 41 considerable rises of temperature with which increase of sickness was associated was 7·82 degrees; the sum of all these rises together being 320·7 degrees. Under average conditions, the amount of increase of sickness which would have occurred in these 41 weeks is 1013 cases. But the actual increase which occurred was 1785 cases, leaving thus 772 cases which must be attributed to the predominant operation of supplementary causes of increase of sickness. Of the 43·5 mean cases of weekly increase in these weeks, then, 24·7 only can be regarded as the result of the rises, while the remaining 18·8 cases must be attributed to the operation of supplementary causes. The combined force of operation of these supplementary causes was then equal to what, under average circumstances, would be exerted by 244·30 degrees of rise of temperature, or by a further mean weekly rise of 5·96 degrees.

#### B. *As to the amount of decrease of sickness.*

1st. The amount of decrease of sickness, when it occurred in association with rises of mean weekly temperature as the result of the predominating operation of antagonistic influences, was controlled by the influence of the rises of temperature.

This appears from observing that the mean amount of weekly decrease of sickness was greater in those weeks in which the rises of temperature were comparatively small than

in those in which they were more extensive. Thus, in the 30 weeks of slight rise in which decrease of sickness occurred the mean amount was 32·8 cases; in the 32 weeks of moderate rise, 23·7 cases; in the 12 weeks of considerable rise, 21·2 cases.

2nd. The amount of the decrease of sickness, when it took place in association with rises of mean weekly temperature, was mainly dependent upon the force exerted by the meteorological conditions antagonistic to the operation of the rises, part of which force was expended in nullifying the influence of the rises of temperature, and part in producing manifest decrease of sickness.

The mean extent of the 74 weekly rises of temperature with which decrease of sickness was associated was 3·14 degrees, the sum of all the rises together being 232·5 degrees. Under ordinary circumstances increase of sickness might have been looked for in the course of these weeks, amounting in the sum to 735 cases, or a mean number of 9·9 cases per week. But no such increase took place; it was nullified by the operation of other predominating antagonistic conditions. A further effect of these antagonistic conditions was to occasion actual manifest decrease of sickness to the amount altogether of 2000 cases, or a mean of 27 cases weekly. The virtual reduction of sickness then that took place in the course of these 74 weeks was 2735 cases, or a mean weekly reduction of 36·9 cases. The entire value of the force of the conditions thus reducing the sickness in these weeks may be expressed by saying that it was equal to what, under average conditions, would be exerted by 865·5 degrees of fall of temperature, or a mean weekly reduction of temperature to the extent of 11·7 degrees. Of this force, a force equal to 633 degrees, or a mean force equal to 8·56 degrees of fall per week, was expended in producing manifest decrease of sickness after the influence of the rises was nullified.

*As to slight rises.*—The mean extent of the 30 slight rises of temperature with which decrease of sickness was associated was 1·05 degrees, the sum of all the rises together being 31·7 degrees. Hence, under ordinary circumstances, an

increase of sickness, to the total amount of 100 cases, or a mean weekly increase of 3·3 cases, would have occurred. This increase was nullified, and a manifest decrease of sickness produced, to the amount altogether of 984 cases or 32·8 cases weekly. The virtual reduction of sickness, then, that took place in the course of these 30 weeks was 1084 cases, or a mean weekly reduction of 36·1 cases. The entire value of the force of the reducing conditions, was then equal to that of 343·03 degrees of fall of temperature, or of a mean weekly fall to the extent of 11·43 degrees. Of this force, after nullifying the rises, a force equal to 311·33 degrees, or a mean weekly force equal to 10·38 degrees of fall, was expended in producing manifest decrease of sickness.

*As to moderate rises.*—The mean extent of the 32 moderate rises of mean temperature with which decrease of sickness was associated was 3·24 degrees—the sum of all the rises together being 103·7 degrees. Hence, under average conditions, an increase of sickness to the total amount of 328 cases, or a mean weekly increase of 10·2 cases, would have occurred. This increase was nullified and a manifest decrease of sickness produced, amounting altogether to 761 cases, or 23·7 cases weekly. The virtual reduction of sickness then that took place in the course of these 32 weeks was 1089 cases, or a mean weekly reduction of 33·9 cases. The entire value of the force of the reducing conditions was then equal to that of a fall of 344·62 degrees, or of a mean weekly fall of 10·77 cases. Of this force, after nullifying the rises, a force equal to 240·92 degrees, or a mean weekly force of 7·53 degrees, was expended in producing manifest decrease of sickness.

*As to considerable rises.*—The mean extent of the 12 considerable rises of temperature with which decrease of sickness was associated was 8·09 degrees, the sum of all the rises together being 97·1 degrees. Hence, under average conditions, an increase of sickness to the total amount of 307 cases, or a mean weekly increase of 25·6 cases, would have occurred. This increase was nullified, and a manifest decrease of sickness produced, amounting altogether to 255 cases, or 21·2 cases weekly. The virtual reduction of sickness then that took

place in the course of these 12 weeks was 562 cases, or a mean weekly decrease of 46·8 cases. The entire value of the force of the reducing conditions was then equal to that of a fall of 177·85 degrees, or of a mean weekly fall of 14·82 degrees. Of this force, after nullifying the rises, a force equal to 80·74 degrees, or a mean weekly force equal to 6·73 degrees of fall of temperature, was expended in producing manifest decrease of sickness.

There is upon the table one week in which neither increase nor decrease of sickness occurred. The mean rise of temperature in this week was 3·5 degrees, corresponding, under average circumstances, to an increase of 11 cases of sickness. The increase was simply nullified by the operation of predominating causes of decrease of sickness of similar force to the rise of temperature.

## 2. *Falls.*

The mean amount of decrease of sickness in the weeks of fall of mean temperature in which decrease took place was greater than that of increase of sickness in the weeks in which increase took place.

Thus the total amount of decrease of sickness in the 113 weeks in which it occurred was 4538 cases—mean, 40·2 cases; and the total amount of increase of sickness in the 76 weeks in which it occurred was 2253 cases—mean, 29·6 cases.

The influence of the falls of temperature in producing the decrease of sickness that was observed, and in checking the operation of the causes of increase was shown by the fact that the mean weekly amount of all the weekly decreases which occurred in the course of the 389 weeks was 34·96 cases; and of all the increases 32·99.

### A. *As to the amount of decrease of sickness.*

1st. The amount of decrease of sickness when it occurred was very much governed by the extent of the falls of mean temperature with which it was associated.



This appears from observing that the mean weekly amount of decrease of sickness was greater in those weeks in which the extent of the falls of temperature was great than those in which it was comparatively small. Thus :

In the 29 weeks of slight fall in which decrease of sickness occurred the mean amount of the decrease was 33.1 cases ; in the 52 weeks of moderate fall, 35.1 cases ; in the 32 weeks of considerable fall, 54.8 cases.

2nd. The amount of decrease of sickness also was to a certain degree dependent upon other supplementary meteorological conditions.

The mean extent of the 113 falls of mean weekly temperature with which decrease of sickness was associated was 3.86 degrees, the sum of all the falls together being 436.3 degrees. Under average circumstances the amount of decrease of sickness that might have been anticipated from falls to this extent is 1379 cases. But the amount of decrease of sickness that actually occurred in these 113 weeks was 4538 cases, leaving thus 3159 cases of decrease to be accounted for by the predominant operation in these weeks of supplementary causes of decreased sickness. Of the 40.2 mean weekly cases of decrease then, only 12.2 cases can be regarded as due to the falls of temperature, the remainder, 28 cases, being attributable to supplementary conditions. The combined force of these supplementary conditions was then equal to what, under average circumstances, would be exerted by 999.68 degrees of fall of temperature, or by a further mean weekly fall of 8.85 degrees.

*As to slight falls.*—The mean extent of the 29 slight falls of mean weekly temperature with which decrease of sickness was associated was 1.07 degrees, the sum of all the falls together being 31.1 degrees. Such an extent of falls would correspond, under average circumstances, to a decrease of sickness amounting altogether to 98 cases. But the actual decrease that occurred in the course of these 29 weeks was 960 cases, leaving thus 862 cases to be accounted for by the predominant operation of supplementary causes of decrease. Of the 33.1 mean weekly cases of decrease then only 3.4

cases can be regarded as due to the slight falls of temperature, while the remainder, 297 cases, must be referred to the operation of supplementary conditions. The combined force of these supplementary conditions was then equal to what, under average circumstances, would be exerted by 272.78 degrees of fall of temperature, or by a further mean weekly fall of 9.40 degrees.

*As to moderate falls.*—The mean extent of the 52 moderate falls of temperature with which decrease of sickness was associated was 3.27 degrees—the sum of all the falls together being 170.4 degrees. Under average circumstances falls to this extent would correspond to a decrease of sickness to the total amount of 533 cases. But the actual sum of the decreases was 1823 cases, leaving thus 1285 cases to be accounted for by the predominant operation of supplementary causes of decrease of sickness. Of the 35.1 mean weekly cases of decrease then only 10.3 cases can be regarded as due to the moderate falls of temperature, while the remainder 24.8 cases must be referred to the operation of supplementary conditions. The combined force of these supplementary conditions then was equal to what, under average circumstances, would be exerted by 406.65 degrees of fall of temperature, or by a further mean weekly fall to the extent of 7.82 degrees.

*As to considerable falls.*—The mean extent of the 32 considerable falls with which decrease of sickness was associated was 7.33 degrees—the sum of all the falls together being 234.8 degrees. Under average circumstances falls to this extent would correspond to a decrease of sickness to the total amount of 742 cases. But the actual sum of the decreases was 1755 cases, leaving thus 1013 cases to be accounted for by the predominant operation of supplementary causes of decrease of sickness. Of the 54.8 mean weekly cases of decrease then only 23.2 cases can be regarded as due to the considerable falls of temperature, while the remaining 31.6 cases must be referred to the operation of supplementary conditions. The combined force of these supplementary conditions then was equal to what, under average circumstances, would be exerted

by 320·57 degrees of fall of temperature, or by a further mean weekly fall to the extent of 10·01 degrees.

*B. As to the amount of increase of sickness.*

1st. The amount of increase of sickness, when it occurred in association with falls of mean weekly temperature as the result of the predominant operation of antagonistic influences, was controlled by the influence of the falls of temperature.

This appears from observing that the mean amount of weekly increase of sickness was greater in those weeks in which the falls of temperature were comparatively small than in those in which they were more extensive. Thus:

In the 26 weeks of slight fall in which increase of sickness occurred the mean amount was 39·5 cases; in the 34 weeks of moderate fall, 25·1; in the 16 weeks of considerable fall, 23·1.

2nd. The amount of the increase of sickness, when it took place in association with falls of mean weekly temperature, was mainly dependent upon the force exerted by the meteorological conditions antagonistic to the operation of the falls, part of which force was expended in nullifying the influence of the falls of temperature, and part in producing manifest increase of sickness.

The mean extent of the 76 weekly falls of temperature with which increase of sickness was associated was 3·43 degrees—the sum of all the falls together being 260·7 degrees. Under ordinary circumstances decrease of sickness might have been looked for in these weeks to the total amount of 824 cases, or a mean weekly decrease of 10·8 cases. But no such decrease took place; it was nullified by the operation of other predominating antagonistic conditions. A further effect of these antagonistic conditions was to occasion manifest increase of sickness to the amount altogether of 2253 cases, or a mean of 29·6 cases weekly. The virtual increase of sickness then that took place in the course of these 76 weeks was 3077 cases, or a mean weekly increase of 40·5 cases. The entire value of the force of the conditions thus increasing the sickness in these weeks may be expressed by saying that it was equal to what, under average conditions, would be exerted by 973·73 degrees of rise of temperature, or a mean weekly rise

to the extent of 12·81 degrees. Of this force, after nullifying the falls, a force equal to 713·03 degrees of rise of temperature, or a mean weekly force equal to 9·38 degrees of rise was expended in producing manifest increase of sickness.

*As to slight falls.*—The mean extent of the 26 slight falls of temperature with which increase of sickness was associated was 1·07 degrees—the sum of all the falls together being 27·9 degrees. Hence, under ordinary circumstances, we might have looked for decrease of sickness in these weeks to the amount of 88 cases, or a mean weekly decrease of 3·4 cases. This decrease was nullified, and a manifest increase of sickness occasioned the sum of which was 1028 cases, or a weekly mean of 39·5 cases. The virtual increase of sickness then which took place in the course of these 26 weeks was 1116 cases, or a mean weekly increase of 42·9 cases. The entire value of the force of the conditions thus increasing sickness was therefore equal to what, under average circumstances, would be exerted by 353·16 degrees of rise of mean temperature, or a mean weekly rise of 13·58 degrees. Of this force, after nullifying the falls, a force equal to 325·26 degrees of rise of temperature, or a mean weekly force equal to 12·51 degrees, was expended in producing manifest increase of sickness.

*As to moderate falls.*—The mean extent of the 34 moderate falls of temperature with which increase of sickness was associated was 3·45 degrees—the sum of all the falls together being 117·6 degrees. Hence, under ordinary circumstances, we should have looked for decrease of sickness in these weeks to the amount of 372 cases, or a mean weekly decrease of 10·9 cases. The decrease was nullified, and a manifest increase of sickness occasioned, the sum of which was 855 cases, or a weekly mean of 25·1 cases. The virtual increase of sickness then which took place in the course of these 34 weeks was 1227 cases, or a mean weekly increase of 36 cases. The entire value of the force of the conditions thus increasing sickness was then equal to what, under average circumstances, would be exerted by 394·62 degrees of rise of mean temperature, or a mean weekly rise of 11·60 degrees. Of this force, after nullifying the falls, a force equal to 277·02

degrees of rise of temperature, or a mean weekly force equal to 8.15 degrees, was expended in producing manifest increase of sickness.

*As to considerable falls.*—The mean extent of the 16 considerable falls of temperature with which increase of sickness was associated was 7.20 degrees—the sum of all the falls together being 115.2 degrees. Hence, under ordinary circumstances, we should have looked for decrease of sickness in these weeks to the amount of 364 cases, or a mean weekly decrease of 22.7 cases. This decrease was nullified and a manifest increase of sickness occasioned, the sum of which was 370 cases, or a weekly mean of 23.1 cases. The virtual increase of sickness then which took place in the course of these 16 weeks was 734 cases, or a mean weekly increase of 45.9 cases. The entire force of the conditions thus increasing sickness was equal to what, under average circumstances, would be exerted by 232.28 degrees of rise of mean temperature, or a mean weekly rise of 14.54 degrees. Of this force, after nullifying the falls, a force equal to 117.08 degrees of rise of temperature, or a mean weekly force equal to 7.34 degrees, was expended in producing manifest increase of sickness.

There are upon Table 2 four weeks in which neither decrease nor increase of sickness occurred. The mean extent of the falls in these four weeks was 0.7 degrees—the sum of all together being 3.1 degrees. In these weeks the decrease of sickness was simply nullified by the predominant influence of supplementary antagonistic conditions. Had this not been the case decrease of sickness to the amount of 9 cases might have been looked for, or a mean weekly decrease of sickness of 2.4 cases.

Three of these were weeks of slight fall in which the mean extent of the falls was 0.3 degrees—the sum of all together being 1.1 degrees. The decrease was nullified by antagonistic conditions of equal force: otherwise decrease of sickness to the amount of 3 cases, or a weekly decrease of 1.1 cases, might have been looked for.

The remaining week was one of moderate fall to the extent of 2 degrees, the amount of decrease nullified in this week being therefore 6 cases.

The results of the foregoing discussion will be rendered more distinct by placing them in a tabular form, thus :—

Average result of a change of 1 degree mean weekly temperature=a fluctuation of sickness to the amount of 3·16 degrees.

### RISES.

#### *General results of the 196 rises.*

	Number of rises	Total extent in degrees.	Estimated clear gain by way of increase of sickness.	Actual clear increase of sickness.	Number of cases of increase above or below estimate.	Number of degrees of temperature represented by the difference of estimated and actual increase.	
			Cases.	Cases.	Cases.	Weekly mean.	Sum.
Generally .....	196	733·5°	2317	2246	— 71	— 22·47°	— 0·11°
Slight rises .....	70	70·7°	223	182	— 41	— 12·97°	— 0·18°
Moderate rises ...	73	245·0°	774	534	— 240	— 75·95°	— 1·04°
Considerable rises	53	417·8°	1320	1530	+ 210	+ 66·46°	+ 1·25°

*Assumed frequency with which increase and decrease of sickness occurred as the result of the operation of rises and of supplementary conditions.*

	Weeks of increase of sickness, 61·73 per cent.				Weeks of decrease or no increase of sickness, 38·27 per cent.			
	Increase due to joint operation of rises and supplementary causes of increase.		Increase due to rises alone overcoming antagonistic supplementary causes.		Decrease due to antagonistic supplementary causes.		Increase nullified by antagonistic supplementary causes.	
	Number of weeks.	Per cent. of weeks of rise.	Number of weeks.	Per cent. of weeks of rise.	Number of weeks.	Per cent. of weeks of rise.	Number of weeks.	Per cent. of weeks of rise.
Rises generally...	102	52·00	19	9·73	74	37·75	1	0·52
Slight rises .....	36	52·00	4	5·14	30	42·86		
Moderate rises ...	38	52·00	2	2·79	32	43·84	1	1·37
Considerable rises	28	52·00	13	25·36	12	22·64		

*Amount of Increase of Sickness when Increase occurred.*

Rises.	Number of weeks of increased sickness.	Extent of rises.		Value in degrees of rise of temperature of supplementary causes of increase in predominant operation.		Value in degrees of rise of temperature of united causes of increase of sickness.		Estimated increase of sickness.		Amount of increase due to supplementary causes.		Joint results of rises of temperature and supplementary causes of increase.	
		Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.
Generally .....	121	497°5'	4°11'	846°20'	6°99'	1843°70'	11°10'	1572	12°99	2674	22°10	4246	35°09
Slight .....	40	39°0'	0°97'	330°06'	8°25'	369°06'	9°22'	123	3°1	1043	26°0	1166	29°1
Moderate .....	40	137°8'	3°44'	272°15'	6°80'	409°95'	10°24'	435	10°9	860	21°4	1295	32°3
Considerable ...	41	320°7'	7°82'	244°30'	5°96'	565°00'	13°78'	1013	24°7	772	18°8	1785	43°5

*Amount of Decrease of Sickness when Decrease occurred.*

Rises.	Number of weeks of decreased sickness.	Extent of rises.		Value in degrees of fall of temperature of supplementary causes of decrease in predominant operation producing manifest decrease of sickness.		Value in degrees of fall of temperature of supplementary causes of decrease in predominant operation producing manifest decrease of sickness.		Estimated normal increase of sickness nullified.		Manifest decrease of sickness produced.		Virtual decrease of sickness.	
		Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.
Generally .....	74	232.5°	3.14°	633.0°	8.56°	865.50°	11.70°	735	9.9	2000	27.0	2735	36.9
Slight .....	30	31.7°	1.06°	311.33°	10.38°	343.03°	11.43°	100	3.3	984	32.8	1084	36.1
Moderate .....	32	108.7°	3.24°	240.92°	7.53°	344.62°	10.77°	328	10.2	761	23.7	1089	33.9
Considerable...	12	97.1°	8.09°	80.75°	6.73°	177.85°	14.82°	307	25.6	255	21.2	562	46.8



*Summary.*—1. In the majority of the 196 weeks in which a rise of mean temperature above that of the previous week occurred, the rise of temperature was associated with an increase of general sickness. The general result of the inquiry is that increase of sickness is the normal effect of a rise of temperature.

2. In the remaining weeks (with one exception) decrease of sickness took place. In the exceptional week no alteration in the amount of sickness was noted. Decrease of sickness or the non-occurrence of increase of sickness in association with a rise of temperature must be regarded as due to the predominant operation of antagonistic meteorological conditions either antecedent to or concurrent with the rises; their force of operation may be expressed in a number of degrees of fall of mean temperature to which they are equivalent in operation.

3. Increase of sickness was, on the whole, observed to occur more frequently in the weeks in which the extent of the rise of temperature was great than when it was comparatively small.

4. The occurrence of increase or decrease of sickness in any individual week of rise of mean temperature depends partly upon the efficient force of the rise (its extent) overcoming or being overcome by antagonistic influences, and partly upon the predominance of operation and force of operation of other influences (supplementary causes of increase or decrease of sickness) co-operating with the rises or antagonising them.

5. Notwithstanding the probable co-operation of other causes of increased sickness in 102 out of the 196 weeks of rise of temperature, the clear gain by way of increase of sickness, in the course of these 196 weeks, did not quite reach the amount which might have been anticipated from a comparison of the extent of change of temperature generally in the 389 weeks tabulated, and the amount of clear gain by way of increase or decrease of sickness which those weeks furnished.

6. Dividing the weeks according as the rise of temperature was slight, moderate, or considerable, it was found that the

deficiency of increase of sickness referred to was only observed in the weeks in which the rises were slight or moderate, and that in the latter especially the clear increase of sickness was most deficient in amount. In the weeks of considerable rise, on the contrary, the clear gain by way of increase of sickness was in excess of what might have been anticipated.

7. The excess or deficiency of the final gain by way of increase of sickness in the weeks of rises of temperature must be held to be dependent upon the force of operation of those supplementary meteorological conditions which assist or antagonise the operations of rises of temperature. Hence it may be inferred that, in the weeks of rise of temperature taken altogether, the force of the antagonistic conditions was such as to reduce the increase of sickness below what would have resulted had not these antagonistic influences predominated in the course of the 196 weeks: that in the 70 weeks of slight rise of temperature the antagonistic conditions predominated sufficiently to reduce the final gain of increased sickness to what it might have been expected to be if the mean extent of the rises had been 0.18 degrees less than the actual extent: that in the 73 weeks of moderate rise the predominance of the antagonistic influences referred to was much more marked, the result being a lessened gain by way of increase such as would have been looked for had the mean weekly extent of the rise been 1.04 degrees less than the actual extent: that on the contrary in the 53 weeks of considerable rise the co-operating influences predominated in force in such a manner as to augment the increase beyond what might have been anticipated, and making the clear gain of increase of sickness equal to that which might have been looked for had the mean extent of the rises been 1.25 degrees greater than the actual extent.

8. The mean amount of increase of sickness in those weeks in which increase of sickness took place was greater than that of decrease of sickness in the weeks in which decrease took place.

9. The mean amount of increase of sickness in the weeks in which it occurred was greater the greater the extent of the

rises of temperature with which it was associated. Thus, it was greater in the weeks of moderate than of slight rise of temperature, and greater in the weeks of considerable than of moderate rise.

10. In those weeks in which increase of sickness took place in association with rise of temperature of all degrees of extent, the greater part of the increase (2674 out of 4246 cases) was due to the influence exerted by other predominating supplementary causes assisting the operation of the rises.

11. In those weeks of slight rise of temperature in which increase of sickness took place, this operation of supplementary causes of increase of sickness was most marked. Out of 1166 cases by which sickness was increased, 1043 cases resulted from the predominating influence of these supplementary conditions.

12. In those weeks of moderate rise of temperature in which increase of sickness took place, the amount of increase attributable to the supplementary conditions promotive of increase of sickness was still disproportionately large. Out of 1295 cases by which sickness was increased in these weeks, 860 cases resulted from the predominant operation of these conditions.

13. In those weeks of considerable rise of temperature in which increase of sickness took place, although the operation of the supplementary causes of increase was still marked, yet it was not in excess of the operation of the rises themselves. Out of 1785 cases by which sickness was increased in these weeks, 772 resulted from the predominant operation of these conditions, while 1013 were due to the influence of the rises.

14. Thus, according as the extent of the rises with which increase of sickness was associated was greater, so the share taken by the rises in producing the increase was also greater, and the share taken by the other supplementary conditions promotive of increase was less.

15. The mean amount of decrease of sickness when it occurred in association with rises of temperature was less in the weeks in which the rises of temperature were more extensive, and greater in those weeks in which they were less

extensive. Thus, it was greatest in weeks when it was associated with slight rises, less when associated with moderate rises, and least when associated with considerable rises.

16. In estimating the force of the conditions producing decrease of sickness in spite of the tendency to increase imparted by a rise of temperature, it is necessary to take into account not only the manifest decrease produced, but the neutralisation of the influence of the associated rises also.

17. In the 74 weeks in which decrease of sickness occurred with rises of temperature of various degrees of extent, the amount of free and predominant antagonistic force expended in producing manifest decrease of sickness was much greater than that expended in neutralising the rises, the proportion of the former to the latter being as  $633^{\circ}$  to  $232^{\circ}5$ .

18. In the 30 weeks in which decrease of sickness occurred with slight rises of temperature, the disproportion referred to was most marked, the amount of antagonistic force expended in producing manifest decrease being to that expended in neutralising the rises as  $311^{\circ}33$  to  $31^{\circ}7$ .

19. In the 32 weeks in which decrease of sickness occurred with moderate rises of temperature, the amount of antagonistic force expended in producing manifest decrease of sickness was to that expended in neutralising the rises as  $240^{\circ}92$  to  $103^{\circ}7$ .

20. In the 12 weeks in which decrease of sickness occurred with considerable rises of temperature, the amount of antagonistic force expended in neutralising the rises was on the contrary rather greater than that expended in producing manifest decrease of sickness, the latter being to the former as  $80^{\circ}75$  to  $97^{\circ}1$ .

21. Thus, the greater the extent of the rises with which decrease of sickness was associated, the greater was the proportion of the antagonistic force expended in neutralising the rises, and the less that expended in producing manifest decrease of sickness.

22. On the whole, the mean weekly amount of antagonistic force expended in neutralising the rises and producing manifest decrease of sickness was greater in the weeks of considerable rise in which decrease occurred than in those of

slight or moderate rise, and rather greater in the weeks of slight than in those of moderate rise.

23. Comparing now the weeks of rise of temperature in which sickness was increased with those in which it was decreased we find that the mean weekly value of the forces of decrease neutralising the rises and producing manifest decrease in the latter set of weeks was very slightly greater (by  $\cdot 6$  degrees) than that of all the forces of increase combined and operating in the former set of weeks. A similar observation applies to the weeks of slight, moderate, and considerable rise respectively, in which increase and decrease of sickness took place, the difference being most marked in the weeks of slight rise.

24. Comparing the force of the predominating supplementary conditions promoting increase of sickness in the 121 weeks in which sickness was increased with the force of the predominating supplementary conditions neutralising the rises and producing decrease of sickness in the 74 weeks of decreased sickness, we find that the sum of the forces of the supplementary conditions of promoting decrease of sickness was rather greater than that of the supplementary conditions promoting increase of sickness (viz., as  $865^{\circ}5$  to  $846^{\circ}2$ ), and that the mean weekly force of the former was to that of the latter as  $11^{\circ}7$  to  $6^{\circ}99$ . In the 30 weeks of slight rise in which sickness was decreased the sum of the force of the supplementary causes of decrease was to that of the supplementary causes of increase in the 40 weeks in which sickness was increased as  $343^{\circ}03$  to  $330^{\circ}06$ , the difference in the two sums not being great; the mean weekly force of the former was, however, to that of the latter as  $11^{\circ}43$  to  $8^{\circ}25$ . In the 32 weeks of moderate rise in which sickness was decreased the sum of the forces of the supplementary causes of decrease was much greater than that of the supplementary causes of increase in the 40 weeks of moderate rise on which sickness was increased (viz., as  $344^{\circ}62$  to  $272^{\circ}15$ ) the mean weekly force of the former being also to that of the latter as  $10^{\circ}77$  to  $6^{\circ}8$ . In the 12 weeks of considerable rise, however, in which sickness was decreased, the sum of the force of the supplementary causes of decrease in operation was much

less than that of the supplementary causes of increase of sickness in the 41 weeks of considerable rise in which sickness was increased (viz., as  $177^{\circ}\cdot85$  to  $244^{\circ}\cdot3$ ); but taking the number of weeks into account, the mean weekly force of the former was to that of the latter as  $14^{\circ}\cdot82$  to  $5^{\circ}\cdot96$ .

### FALLS.

#### *General results of the 193 falls.*

	Number of falls.	Total extent in degrees.	Estimated clear gain by way of decrease of sickness.	Actual clear decrease of sickness.	Number of cases of decrease above or below estimate.	Number of degrees of temperature represented by the difference of estimated and actual decrease.	
			Cases.		Cases.	Sum.	Weekly mean.
Generally .....	193	$700\cdot1^{\circ}$	2212	2285°	+ 73	+ $23\cdot00^{\circ}$	+ $0\cdot12^{\circ}$
Slight .....	58	$60\cdot1^{\circ}$	190	—	— 258	— $81\cdot65^{\circ}$	— $1\cdot40^{\circ}$
Moderate .....	87	$290\cdot0^{\circ}$	916	968	52	+ $16\cdot45^{\circ}$	+ $0\cdot18^{\circ}$
Considerable .....	48	$350\cdot0^{\circ}$	1106	1385	+ 279	+ $88\cdot29^{\circ}$	+ $1\cdot84^{\circ}$

*Assumed frequency with which decrease and increase of sickness occurred, as the result of the operation of falls and of the supplementary conditions.*

	Weeks of decrease of sickness, 58·55 per cent.				Weeks of increase of sickness or no decrease, 41·45 per cent.			
	Decrease due to joint operation of falls and supplementary causes of decrease.		Decrease due to falls alone overcoming antagonistic supplementary causes.		Increase due to antagonistic supplementary causes.		Decrease nullified by antagonistic supplementary causes.	
	Number of weeks.	Per cent. of weeks of fall.	Number of weeks.	Per cent. of weeks of fall.	Number of weeks.	Per cent. of weeks of fall.	Number of weeks.	Per cent. of weeks of fall.
Falls generally ..	93	48·00	20	10·55	76	39·38	4	2·07
Slight falls .....	28	48·00	1	2·00	26	44·83	3	5·17
Moderate falls ...	42	48·00	10	11·77	34	39·08	1	1·15
Considerable falls	23	48·00	9	18·67	16	33·33		

*Amount of decrease of sickness when decrease occurred.*

	Number of weeks of decreased sickness.	Extent of falls.		Value in degrees of fall of temperature of supplementary causes of decrease in pre-dominant operation.		Value in degrees of fall of temperature of united causes of decrease of sickness.		Estimated decrease of sickness.		Amount of decrease due to supplementary causes.		Joint result of falls of temperature of supplementary causes of decrease.	
		Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.
Falls.													
Generally .....	113	436.3°	3.86°	999.68°	8.86°	1435.98°	12.71°	1379	12.2	3159	28.0	4538	40.2
Slight .....	29	31.1°	1.07°	272.78°	9.40°	303.88°	10.47°	98	3.4	862	29.7	960	33.1
Moderate ... ..	52	170.4°	3.27°	406.65°	7.82°	577.05°	11.09°	538	10.3	1285	24.8	1823	35.1
Considerable ...	32	234.8°	7.33°	320.57°	10.01°	555.37°	17.34°	742	23.2	1013	31.6	1755	54.8

*Amount of increase of sickness when increase occurred.*

	Number of weeks of increased sickness.	Extent of fall.		Value in degrees of rise of temperature of supplementary causes of increase in predominant operation producing manifest increase of sickness.		Value in degrees of rise of temperature of supplementary causes of increase in predom. modifying falls and producing manifest increase of sickness.		Estimated normal decrease of sickness nullified.		Manifest increase of sickness produced.		Virtual increase of sickness.	
		Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.	Sum.	Mean.
Falls.													
Generally .....	76	260.7°	3.43°	713.03°	9.38°	973.73°	12.81°	824	10.8	2853	29.6	3077	40.5
Slight .....	26	27.9°	1.07°	825.26°	12.51°	353.16°	13.58°	88	3.4	1028	39.5	1116	42.9
Moderate .....	34	117.6°	3.45°	277.02°	8.15°	394.62°	11.60°	372	10.9	855	25.1	1227	36.0
Considerable ...	16	115.2°	7.20°	117.08°	7.34°	232.23°	14.54°	364	22.7	370	23.1	734	45.9



*Summary.*—1. In the majority of the 193 weeks in which a fall of mean temperature below that of the previous week occurred, the fall was associated with a decrease of general sickness. The general result of the inquiry is, that decrease of sickness is the normal effect of a fall of temperature.

2. In the remaining weeks (with four exceptions) increase of sickness took place. In the exceptional weeks no alteration in the amount of general sickness was noted. Increase of sickness or the non-occurrence of decrease of sickness in association with a fall of temperature must be regarded as due to the predominant operation of antagonistic meteorological conditions either antecedent to or concurrent with the falls; their force of operation may be expressed in a number of degrees of rise of mean temperature to which they are equivalent in operation.

3. Decrease of sickness on the whole was observed to occur more frequently in the weeks in which the extent of the fall of temperature was great than when it was comparatively small.

4. The occurrence of decrease or increase of sickness in any individual week of fall of mean temperature depends partly upon the efficient force of the fall (its extent) overcoming or being overcome by antagonistic influences, and partly upon the predominance of operation and force of operation of other influences (supplementary causes of decrease or increase of sickness) co-operating with the falls or antagonising them.

5. The clear gain by way of decrease of sickness in the course of the 193 weeks of fall exceeded the amount which might have been anticipated from a comparison of the extent of changes of temperature generally in the 389 weeks tabulated, and the amount of clear gain by way of increase or decrease of sickness which those weeks furnished. This excess was nearly to the same amount as the defect in the case of the rises, and it was observed notwithstanding that the co-operation of supplementary causes was probably less frequent than in the case of the rises tabulated.

6. Dividing the weeks according as the fall of temperature was slight, moderate, or considerable, it was found that while there was an excess of decrease of sickness in the weeks of moderate and considerable fall, especially marked in the latter, it was quite otherwise in the weeks of slight fall of temperature. In the weeks of slight fall, taken altogether, it was found that the cases of increase of sickness that took place in the course of them were really more numerous than the cases of decrease of sickness, so that the final result was that in these weeks the balance of fluctuation of sickness was in favour of increase and not of decrease of sickness.

7. The excess or deficiency of the final gain, by way of decrease of sickness in the weeks of fall of temperature, must be held to be dependent upon the force of operation of those supplementary meteorological conditions which assist or antagonise the operation of falls of temperature. Hence it may be inferred that in the weeks of fall of temperature taken altogether, the force of the co-operating conditions was such as to raise the amount of decrease of sickness above what would have resulted, had not these co-operating influences predominated in the course of the 193 weeks: that in the 58 weeks of slight fall of temperature, supplementary antagonistic conditions predominated to such an extent as to produce a final result of increase of sickness similar to what might have been anticipated under average circumstances, had no falls of temperature at all taken place, but in lieu thereof a weekly rise to the mean extent of  $0^{\circ}12$ : that in the 87 weeks of moderate fall of temperature co-operating supplementary influences predominated to such an extent as to augment the decrease of sickness in a manner similar to that which would have resulted from a further mean weekly fall of  $0^{\circ}18$ : and that in the 48 weeks of considerable fall co-operating supplementary influences predominated to such an extent as to augment the decrease in a manner similar to what might have been anticipated from a further mean weekly fall of  $1^{\circ}84$ .

8. The mean amount of decrease of sickness in those weeks in which decrease of sickness took place was greater than

that of increase in the weeks in which increase of sickness took place.

9. The mean amount of decrease of sickness in the weeks in which it occurred was greater the greater the extent of the falls of temperature with which it was associated. Thus it was greater in the weeks of moderate than in those of slight fall of temperature, and greater in the weeks of considerable than in those of moderate fall.

10. In those weeks in which decrease of sickness took place in association with falls of temperature of all degrees of extent, by far the greater part of the decrease (3159 out of 4538 cases) was due to the influence exerted by other predominating supplementary causes assisting the operation of the falls.

11. In those weeks of slight fall of temperature in which decrease of sickness took place, the operation of supplementary causes of decrease was most marked. Out of 960 cases by which sickness was decreased, 862 cases resulted from the predominating influence of these supplementary conditions.

12. In those weeks of moderate fall of temperature in which decrease of sickness took place, the amount of decrease attributable to the supplementary conditions productive of decrease of sickness was still disproportionately large. Out of 1823 cases by which sickness was decreased in these weeks, 1285 resulted from the predominant operation of these conditions.

13. In those weeks of considerable fall of temperature in which decrease of sickness took place, the amount of decrease attributable to the supplementary conditions productive of decrease of sickness was still disproportionately large. Out of 1755 cases by which sickness was decreased in these weeks, 1018 resulted from the predominant operation of these conditions.

14. Thus, according as the extent of the falls with which decrease of sickness associated was greater, so the share taken by the falls in producing the decrease was also greater, and the share taken by the other supplementary conditions promotive of decrease was less.

15. The mean amount of increase of sickness when it occurred in association with falls of temperature was less in the weeks in which the falls of temperature were more extensive and greater in those weeks in which they were less extensive. Thus, it was greatest in the weeks in which it was associated with slight falls, less when associated with moderate falls, and least when associated with considerable falls.

16. In estimating the force of the conditions producing increase of sickness in spite of the tendency to decrease imparted by a fall of temperature, it is necessary to take into account not only the manifest increase produced, but the neutralisation of the influence of the associated falls also.

17. In the 76 weeks in which increase of sickness occurred with falls of temperature of various degrees of extent, the amount of free and predominant antagonistic force expended in producing manifest increase of sickness was much greater than that expended in neutralising the falls—the proportion of the former to the latter being as  $713^{\circ}03$  to  $260^{\circ}7$ .

18. In the 26 weeks in which increase of sickness occurred with slight falls of temperature, the disproportion referred to was most marked, the amount of antagonistic force expended in producing manifest increase being to that expended in neutralising the falls as  $325^{\circ}26$  to  $27^{\circ}9$ .

19. In the 34 weeks in which increase of sickness occurred with moderate falls of temperature, the amount of antagonistic force expended in producing manifest increase of sickness, was to that expended in neutralising the falls  $277^{\circ}02^{\circ}$  to  $117^{\circ}6^{\circ}$ .

20. In the 16 weeks in which the increase of sickness occurred with considerable falls of temperature, the amount of antagonising force expended in producing manifest increase of sickness, was nearly equal to that expended in neutralising the falls, the former being to the latter as  $117^{\circ}08$  to  $115^{\circ}2$

21. Thus, the greater the extent of the falls with which increase of sickness was associated, the greater was the proportion of antagonistic force expended in neutralising the

falls, and the less that expended in producing manifest increase of sickness.

22. On the whole, the mean weekly amount of antagonistic force expended in neutralising the falls and producing manifest increase of sickness, was greater in the weeks of considerable fall in which increase occurred, than in those of slight or moderate fall, and greater in the weeks of slight than of moderate fall.

23. Comparing now the weeks of fall of temperature in which sickness was decreased with those in which it was increased, we find that the mean weekly value of the forces of increase neutralising the falls and producing manifest increase in the latter set of weeks was nearly the same as that of all the forces of increase combined, and operating in the former set of weeks: that in the weeks of slight fall the mean weekly force of the causes of increase when it occurred was much greater than that of the causes of decrease when decrease occurred: that in the weeks of moderate fall the force of the causes of increase when it occurred was slightly greater only than that of the causes of decrease when decrease occurred: but, on the contrary, that in the weeks of considerable fall in which decrease occurred the mean weekly force of the causes of decrease combined was much greater than that of the causes of increase when increase occurred.

24. Comparing the force of the predominating supplementary condition promoting decrease of sickness in the 113 weeks in which sickness was decreased with the force of the predominating supplementary conditions neutralising the falls and producing increase of sickness in the 76 weeks of decreased sickness, we find that the sum of the force of the supplementary conditions promoting decrease of sickness was rather greater than that of the supplementary conditions promoting increase of sickness, viz., as  $999^{\circ}68$  to  $973^{\circ}73$ ; but on the other hand, that the mean weekly force of the latter was to that of the former as  $12^{\circ}81$  to  $8^{\circ}85$ . In the 26 weeks of slight fall in which sickness was increased the sum of the forces of the supplementary conditions promoting increase was to that of the supplementary conditions promoting

decrease in the 29 weeks in which sickness was decreased, as  $353^{\circ}16$  to  $272^{\circ}78$ ; and also the mean weekly force of the former was greater than that of the latter, viz., as  $13^{\circ}58$  to  $9^{\circ}40$ . In the 52 weeks of moderate fall in which sickness was decreased the sum of the force of the supplementary conditions promoting decrease was to that of the supplementary conditions promoting increase as  $406^{\circ}65$  to  $394^{\circ}62$ ; but the mean weekly force of the latter was very much greater than of the former, viz., as  $11^{\circ}60$  to  $7^{\circ}82$ . In the 32 weeks of considerable fall in which sickness was decreased the sum of the forces of the supplementary conditions promoting decrease was to that of the supplementary conditions promoting increase as  $302^{\circ}57$  to  $232^{\circ}28$ ; but again, the mean weekly force of the latter was very much greater than of the former, viz., as  $14^{\circ}54$  to  $10^{\circ}01$ .

ON THE SUPPLEMENTARY CONDITIONS GENERALLY CO-OPERATING WITH, OR ANTAGONISING THE INFLUENCE OF THE RISES AND FALLS OF MEAN WEEKLY TEMPERATURE.

It may not be out of place here to gather up a few further deductions arising out of the preceding inquiry, respecting the frequency and force of operation of the supplementary meteorological conditions so often mentioned as modifying the results of the rises and falls of mean temperature in the 889 weeks which have been submitted to discussion. I propose to group them all together, leaving to a future time the inquiry into the influence exerted by each severally.

The principal of these conditions are, the temperature of the night, the daily range of temperature, variations in the amount of atmospheric humidity, the frequency and amount of rainfall, the direction and force of the wind, and the antecedent occurrence of increase or decrease of sickness depending either upon changes of temperature or other causes. These that I have enumerated have appeared to me to be the most important conditions affecting public health. I do not absolutely exclude the operation of changes of barometric pressure, but I am disposed to think that their connection

with fluctuations of sickness is less direct and independent, nor yet that of electrical states of the atmosphere, the amount of sunlight, and of atmospheric ozone, but the operation of these is less readily defined, and will therefore form no part of my present inquiry.

We may continue to express the force of operation of these supplementary conditions (as I term them for the present) in the number of degrees of rise and fall of mean temperature with which they correspond.

1. *As respects all the 389 weeks of change of temperature upon the tables.*

Supplementary conditions promoting increase and opposing decrease of sickness operated on the whole in the course of these weeks, more frequently than those which tend to promote decrease and oppose increase of sickness. That is to say, putting changes of mean temperature out of the question, the supplementary causes of increase of sickness predominated in force over those of decrease of sickness in a larger proportion of the weeks under analysis than did those of decrease over those of increase of sickness.

The supplementary causes of increase predominated in 52·00 per cent., and decrease 48·00 per cent.

Then as regards the force of their operation when either the one or the other set of causes predominated—

	Sum in deg. of rise.	Weekly mean.
The entire force of operation of the supplementary causes of increase of sickness when co-operating with the 121 rises of temperature .....	846·20°	6·99°
When antagonising 80 falls of temperature.....	976·83°	12·21°
Sum in 201 weeks.....	1823·03°	9·07°
	Degr. of fall.	
The entire force of operation of the supplementary causes of decrease of sickness when antagonising 75 rises of temperature .....	869·00°	11·59°
When co-operating with 113 falls of temperature .....	999·68°	8·85°
Sum in 188 weeks.....	1868·68°	9·94°

	Sum in degs. of rise.	Weekly mean.
The entire force of operation of the supplementary causes of increase of sickness when co-operating with the 40 slight rises of temperature .....	= 330·06°	— 8·25°
When antagonising, 29 slight falls of temperature.....	= 354·26°	— 12·22°
Sum in 69 weeks.....	= 684·32°	— 9·92°
<hr/>		
	Degs. of fall.	
The entire force of operation of the supplementary causes of decrease of sickness when antagonising 30 slight rises of temperature .....	= 343·08°	— 11·43°
When co-operating with 29 slight falls of temperature	= 272·78°	— 9·40°
Sum in 59 weeks.....	= 615·81°	— 10·44°
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Thus, in the weeks of slight change of mean temperature the *sum of the supplementary forces of increase* of sickness was decidedly *greater* than that of the supplementary forces of decrease—but the *mean weekly force of operation of the causes of decrease* when they predominated was rather *greater* than that of the causes of increase.

Comparing the force of operation of the supplementary causes of increase and decrease of sickness as exhibited in the weeks of slight rise with that exhibited in the weeks of slight fall of temperature, we find—

1st. That the sum of the forces of the causes of *increase* of sickness predominating in the 29 weeks of slight fall, and antagonising the falls, was rather greater than that of the causes of increase predominating in the 40 weeks of slight rise and co-operating with the rises; and also that the mean weekly force of the causes of increase engaged in antagonising the falls was greater than that engaged in co-operating with the rises.

2nd. That the sum of the forces of the causes of *decrease* of sickness predominating in the 30 weeks of slight rise and antagonising the rises was decidedly greater than that of the causes of decrease predominating in the 29 weeks of slight fall and co-operating with the falls; and also that the mean weekly force of the causes of decrease engaged in



Thus, while the supplementary forces of increase predominated more frequently than those of decrease, the *sum of the forces of decrease was greater* than that of the forces of increase, and their *mean weekly force* when they did predominate was *greater also*.

Comparing the *force* of operation of the *supplementary causes* of increase and decrease of sickness as exhibited in the *weeks of rise with that exhibited in the weeks of fall* of temperature, we find—

1st. That the sum of the force of the causes of *increase* of sickness predominant and operating in antagonism of the 80 falls of temperature was decidedly greater than that of the causes of increase predominant and operating in assistance of the 121 rises of mean temperature: and moreover, that the mean weekly force of the causes of increase engaged in antagonising the falls was nearly twice as great as that engaged in co-operating with the rises.

2nd. That the sum of the forces of the causes of *decrease* of sickness predominating in the 113 weeks of fall of temperature, and co-operating with the falls was decidedly greater than that of the causes of decrease predominant in the 75 weeks of rises of temperature and antagonising the rises. But that the mean weekly force of the causes of decrease engaged in antagonising the rises was greater than that engaged in co-operating with the falls.

2. *As respects the 128 weeks of slight change of temperature.*

I have assumed that the frequency with which supplementary causes of increase or decrease of sickness predominate is the same in weeks of slight, moderate, and considerable rise and fall of temperature, as in all weeks of change of temperature taken together, so that here and subsequently I have merely the question of force of operation to consider.

	Sum in degs. of rise.	Weekly mean.
The entire force of operation of the supplementary causes of increase of sickness when co-operating with the 40 slight rises of temperature .....	= 330·06°	— 8·25°
When antagonising, 29 slight falls of temperature.....	= 354·26°	— 12·22°
Sum in 69 weeks.....	= 684·32°	— 9·92°
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	Degs. of fall.	
The entire force of operation of the supplementary causes of decrease of sickness when antagonising 80 slight rises of temperature .....	= 343·08°	— 11·43°
When co-operating with 29 slight falls of temperature .....	= 272·78°	— 9·40°
Sum in 59 weeks.....	= 615·81°	— 10·44°

Thus, in the weeks of slight change of mean temperature the *sum of the supplementary forces of increase* of sickness was decidedly *greater* than that of the supplementary forces of decrease—but the *mean weekly force of operation of the causes of decrease* when they predominated was rather *greater* than that of the causes of increase.

Comparing the force of operation of the supplementary causes of increase and decrease of sickness as exhibited in the weeks of slight rise with that exhibited in the weeks of slight fall of temperature, we find—

1st. That the sum of the forces of the causes of *increase* of sickness predominating in the 29 weeks of slight fall, and antagonising the falls, was rather greater than that of the causes of increase predominating in the 40 weeks of slight rise and co-operating with the rises; and also that the mean weekly force of the causes of increase engaged in antagonising the falls was greater than that engaged in co-operating with the rises.

2nd. That the sum of the forces of the causes of *decrease* of sickness predominating in the 30 weeks of slight rise and antagonising the rises was decidedly greater than that of the causes of decrease predominating in the 29 weeks of slight fall and co-operating with the falls; and also that the mean weekly force of the causes of decrease engaged in

antagonising the rises was greater than that engaged in co-operating with the falls.

3. *As respects the 160 weeks of moderate change of temperature.*

	Sum in degra. of rise.	Weekly mean.
The entire force of operation of the supplementary causes of increase of sickness when co-operating with the 40 moderate rises of temperature .....	= 272·15°	— 6·80°
When antagonising 35 moderate falls of temperature...	= 396·62°	— 11·33°
Sum in 75 weeks.....	= 668·77°	— 8·92°
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	Degr. of fall.	
The entire force of operation of the supplementary causes of decrease of sickness when antagonising the 33 moderate rises of temperature .....	= 348·12°	— 10·55°
When co-operating with 52 moderate falls of temperature .....	= 406·65°	— 7·82°
Sum in 85 weeks.....	= 754·77°	— 8·88°

Thus, in the weeks of moderate change of mean temperature the *sum of the supplementary forces of decrease* of sickness was decidedly *greater* than that of the supplementary forces of increase. The *mean weekly force* with which either operated when predominant was, however, *nearly the same*.

Comparing the force of operation of the supplementary causes of increase and decrease of sickness as exhibited in the weeks of moderate rise with that exhibited in the weeks of moderate fall of temperature, we find—

1st. That the sum of the forces of the causes of increase of sickness predominating in the 35 weeks of moderate fall and antagonising the falls was very much greater than that of the causes of increase predominating in the 40 weeks of moderate rise and co-operating with the rises; and moreover, that the mean weekly force of the causes of increase engaged in antagonising the falls was also very much greater than that engaged in co-operating with the rises.

2nd. That the sum of the forces of the causes of decrease of sickness predominating in the 52 weeks of moderate fall of temperature, and co-operating with the falls, was decidedly greater than that of the causes of decrease predominating in the 33 weeks of moderate rise of temperature and antagonising the rises—but that the mean weekly force of the causes of decrease engaged in antagonising the rises was decidedly greater than that engaged in co-operating with the falls.

4. *As respects the 101 weeks of considerable change of temperature.*

	Sum in degra. of rise.	Weekly mean.
The entire force of operation of the supplementary causes of increase of sickness when co-operating with 41 considerable rises of temperature .....	= 244·20°	— 5·96°
When antagonising 16 considerable falls of temperature .....	= 232·28°	— 14·54°
Sum in 57 weeks.....	= 476·58°	— 8·36°
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	Degra. of fall.	
The entire force of operation of the supplementary causes of decrease of sickness when antagonising the 12 considerable rises of temperature .....	= 177·85°	— 14·82°
When co-operating with 32 considerable falls of temperature .....	= 320·57°	— 10·01°
Sum in 44 weeks.....	= 498·42°	— 11·33°

Thus, in the weeks of considerable change of temperature, the sum of the supplementary forces of decrease of sickness was somewhat greater than that of the supplementary forces of increase—and moreover, the mean weekly force of the causes of decrease when they predominated was much greater than that of the causes of increase.

Comparing the force of operation of the supplementary causes of increase and decrease of sickness, as exhibited in the weeks of considerable rise, with that exhibited in the weeks of considerable fall of temperature, we find,—

1st. That the sum of the force of the causes of *increase* of sickness predominating in the 41 weeks of considerable

rise, and co-operating with the rises, was a little greater only than that of the causes of increase predominating in the 16 weeks of considerable fall, and antagonising the falls—but that the mean weekly force of the causes of increase engaged in antagonising the falls, was very much greater than that engaged in co-operating with the rises.

2nd. That the sum of the forces of the causes of *decrease* of sickness predominating in the 32 weeks of considerable fall, and co-operating with the falls, was nearly twice as great as that of the causes of decrease predominating in the 12 weeks of considerable rise, and antagonising the rises—but that the mean weekly force of the causes of decrease engaged in antagonising the rises was decidedly greater than that engaged in co-operating with the falls.

#### DISTRIBUTION OF FORCE OF THE SUPPLEMENTARY CAUSES OF INCREASE AND DECREASE OF SICKNESS.

We may, in the last place, bring together the numbers which show the manner in which the force of the supplementary causes of increase and decrease of sickness appears to have been distributed.

##### 1. *Supplementary causes of increase.*

A. As regards the sum of these forces =  $1823^{\circ}03$  of rise.

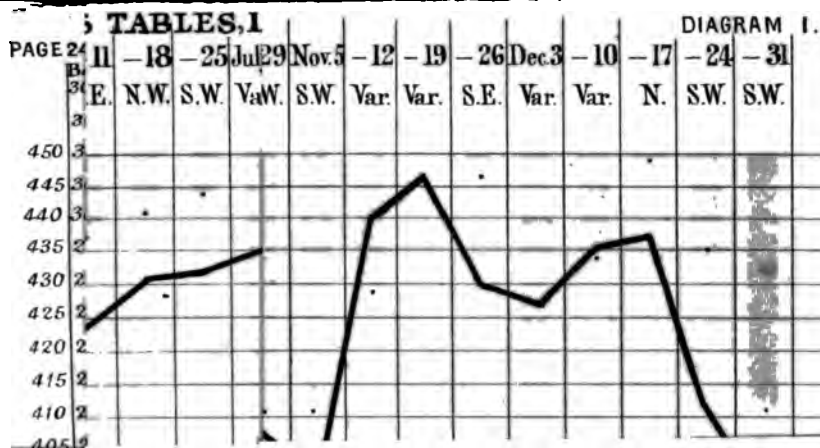
	In degs. of rise.
Engaged in antagonising 16 considerable falls .....	= $232^{\circ}28^{\circ}$
co-operating with 41 considerable rises ...	= $244^{\circ}30^{\circ}$
co-operating with 40 moderate rises .....	= $272^{\circ}15^{\circ}$
co-operating with 40 slight rises .....	= $330^{\circ}06^{\circ}$
antagonising 29 slight falls .....	= $354^{\circ}26^{\circ}$
antagonising 35 moderate falls .....	= $396^{\circ}62^{\circ}$

B. As regards their mean weekly force =  $9^{\circ}07$  of rise.

	In degs. of rise.
When co-operating with considerable rises .....	= $5^{\circ}96^{\circ}$
moderate rises .....	= $6^{\circ}80^{\circ}$
slight rises .....	= $8^{\circ}25^{\circ}$
antagonising moderate falls .....	= $11^{\circ}33^{\circ}$
slight falls .....	= $12^{\circ}22^{\circ}$
considerable falls .....	= $14^{\circ}54^{\circ}$



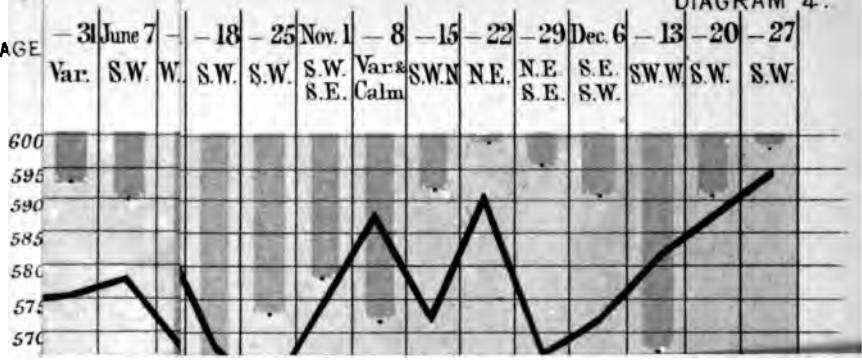








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ON THE INFLUENCE  
OF  
INADEQUATE OPERATIONS ON THE  
THEORY OF CANCER.

BY  
CHARLES H. MOORE, F.R.C.S.,  
VICE-PRESIDENT OF THE SOCIETY, SURGEON TO THE MIDDLESEX,  
AND TO ST. LUKE'S, HOSPITALS.

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THEORIES respecting the nature of Cancer involve the consideration of many facts. An explanation of the disease, to be correct, must reconcile its general relations, such as peculiarities in the family or race, personal habits, and external or geographical condition of the patient, with the various phenomena presented by the disease itself, and with the resemblances and distinctions which exist between it and other tumours. So multitudinous and apparently diverse are the facts on which the rival theories depend that the subject is still shrouded in much obscurity. But much also of the confusion relating to it arises from error and indefiniteness in the observation of the facts themselves.

I do not propose to discuss these general questions in the present paper, but to contribute one of the many series of facts which must concur to their solution. I desire to ascertain if there be a traceable method in the recurrence of

Cancer after operations, or if the renewed disease affect the part continually; to discover if Cancer be solely in its return, conforming to the rule of local functions, or if its morbid material be shed forth into an organ or a region, or into the body as a whole, under an influence which is superior to local conditions.

The bearing of such an inquiry upon the chief theories of Cancer appears to be this:—

There is a theory which includes in the causation of the disease a constitutional malady, something apart from the palpable local tumours, which hovers the phrase is Broca's over the system, ready to alight on any or on many different parts of the body. If the recurrence of Cancer be wholly explicable by conditions in the part which has been the seat of an operation, the addition of another cause is superfluous.

There is again a theory which attributes Cancer to what may be called a textural or an organic diathesis. It is illustrated by the phenomena of many innocent tumours; as when multitudes of villous tumours are strown along a mucous membrane, or neuromas are strung on all the nerves. And it differs from the former theory at least in this, that it restricts the cause of the disease within the organ or texture affected by it. But if it thus limit and localize the cause, this theory likewise connects it with the textural or organic functions, and requires that the tendency to disease should be co-extensive with the respective organ or texture. In the case of double organs therefore, on the removal of one that is cancerous, a prominent liability to the same disease should be transferred to its fellow. I am not prepared to deny that perverted function influences the first outbreak of some Cancers, for there do indistinctly appear to be various causes concerned in setting up similar Cancers, of which causes that may be one. But the theory as a whole may be so far tested by recurrence after operations as to show whether the continued disease belong or not to the organ.

Again, Cancer is sometimes regarded as regional. As so-called supplemental organs are formed in the vicinity of a

principal organic mass, or as some tumours bear a resemblance to the structure near which they originate, so Cancer is held to arise within a region by successive independent growths beyond the limits of the first tumour and of the organ in which it sprang. This theory too may to some extent be tested by the method in which Cancer returns after operations; but when recurrence is late, and not precisely local, the distinction between regional Cancer and a yet more strictly local growth is not easy to draw. It is then necessary to discern between a recurrence traceable to incompleteness of the operation and that return of the disease in which a morbid tendency may be supposed to possess every several structure throughout a region, independently of the extirpated portion which first formed a tumour. No help in solving the question is to be procured by apportioning the irritation of the disease to the blastema or to the cell. It can only be decided by accurate and repeated observations of the mode of recurrence. And there are two elements of uncertainty in the question; uncertainty, namely, whether the operation comprehended all the area over which material from the primary tumour was diffused, and uncertainty respecting the possible duration of inactive life in a fragment of Cancer, severed in the operation from the primary tumour and left in the tissues. When Cancer returns after operations, is it like caries, to which the healthy teeth continue independently liable, though the first diseased tooth have been drawn, or does it correspond to a carious stump, from which only a part of the disease has been taken by breaking off the crown? And, further, if Cancer break out again before the healing of the wound made for its removal, and the recurrence be rightly attributed to incompleteness of the operation, is renewed disease equally traceable to residual fragments when two, five, or a dozen years elapse before recurrence? I may at once state that mere delay is not a sufficient reason for attributing recurrence of Cancer to the outbreak of a new and independent disease in an outlying structure which was healthy at the time of operation. It may be conceived to be quite possible that a residual fragment of the original disease should remain quiescent for years,

since it is known that whole tumours sometimes continue as long without a perceptible alteration of size, and then suddenly enlarge at some one part. On removing such tumours, from the breast or from the parotid region, it is found that the torpid portion has been really cancerous, and probably scirrhus, whilst the character of the newly grown lobe is that of encephaloid ; cells predominating in the recent growth and fibres in the old.

In handling the subject before the Society I have to discuss the adequacy of the operation performed in the several cases which will be cited, and the inferences which should be drawn from the subsequent renewal of the local disease. If, on contemplating a series of such operations, an imputation upon their completeness is thought to be justified, then, so far as they go, they prove it superfluous to invoke the influence of the constitution, or a continuance of local growth which is assumed to persist in spite of a complete removal of the foregoing tumour, in explanation of its recurrence.

For displaying the method in which Cancer returns, it is requisite to select a region which may be readily observed, to distinguish changes in the disease which may be due to functional peculiarities of the part from such as are purely morbid, and, most especially, to take note of the very commencement of the renewed disease, before its early appearances are obliterated. At later periods of the case, when natural textures and scars are destroyed, and glands are extensively invaded, the lessons furnished by the mode of recurrence are lost. The organ most generally convenient for this inquiry is the mamma ; and the conclusions deducible from the recurrence of Cancer in it are strikingly confirmed by observations made in parts which are less within the reach of common notice.

Taught without doubt by foregoing failures, our surgical ancestors adopted a method of operating which might well have been expected to prove effectual against a local recurrence of the disease. They transfixed the base of the mamma, and,

raising it with ligatures, swept off the whole organ, together with all the skin that covered it. The proceeding had a barbarous appearance enough, but it was promising; and, if their knowledge of the disease had led the Surgeons of the time to adopt it before the skin was hopelessly infiltrated, they must have met with more success than they appear to have done. Postponing, however, all operation until the skin was brawny and covered with tubercles, and the deeper textures were involved without limit, they failed too often, with even such extensive cutting as they adopted, to comprehend the entire disease.

It was a mistaken kindness which led to a change of this mode of operating. Under the influence of a clergyman, who expressed what must have been a prevailing horror at such Amazonian surgery, the practice was changed to an incision in the integument, which was reflected in flaps and brought together again after the removal of the cancerous tumour. There could have been no diminution of suffering by this prolongation of the operation, and what was gained by it in neatness was lost in life. With the remains of the breast, as well as in their own texture, the flaps enfolded fragments of diseased substance, and Cancer soon reappeared. It was an operation proper to innocent tumours, which can be removed without the needless mutilation of extirpating the breast, but was quite inapplicable to cases of mammary Cancer.

In our own day various modes of operating are practised. Sometimes the tumour only is removed; sometimes that segment of the breast in which the tumour lies is taken away with it; sometimes, with the intention of extirpating the entire mamma, the failure happens that a portion of it is unwittingly left behind; sometimes the breast is carefully removed, but the propensity of Cancer to extension in the skin is misapprehended, and for symmetry's sake a flap, which even includes the nipple, is preserved; and yet again, there being no definite plan in the mind of the operator but that of cutting wide of the tumour, portions of the organ itself are left behind. The consequence of this last method of operating may be at once apparent when, on examining



the mass thus dug out of the centre of the breast, hard cancerous cords, continuous with the principal tumour, are found to have been cut across. Their outer extremities, prolonged to the margin of the breast, remain behind, and it is fortunate if the discovery be made before the wound is closed and the patient replaced in bed.

By the issue of operations thus conducted opinions on the nature of Cancer are always qualified and sometimes formed. When they have been extensive operations, there is an appearance of reason in assuming them to have been complete. But while, on the one hand, severe operations reduce the patient and favour the growth of Cancer, on the other hand it must be considered that operations are not adequate merely because they have been large. A complete extirpation of Cancer could hardly be looked for except from a large operation for a recent and limited disease. For, whatever be the method of operating, the one important point, both for practice and theory, is to remove the whole. The least remnant is capable of growth, and may spring up into a new tumour with all the energy of the first. Recent experiments with acetic acid well illustrate the tenacity of life and the reproductive power of the minutest fragment of Cancer. By injections of that acid I have repeatedly, in the course of a few weeks, obtained the completest dissolution of a subcutaneous cancerous nodule; yet a few weeks later, in the place where, with the most careful touch, I could detect no remnant of the previous tumour, a growth has begun again, which in time became a nodule like the first. So subtle is the disease against which surgeons have to contend in their operations, and respecting which theories are framed out of its usual recurrence when removed.

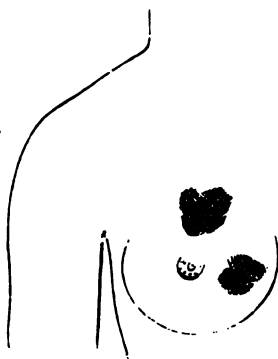
This point appears to me a most important one, and one which would of itself suffice to excuse my thus occupying the attention of the Society. It is sometimes supposed that operations as at present conducted must be regarded as effectual, and that the recurrences so commonly ensuing cannot but be attributed to the nature of the disease. I venture however to aver that the difficulties of completely extirpating Cancer

are much underrated by those who hesitate to suspect the perfectness of our operations. There is too often reason, without calling in the evidence of the microscope, to expect a return of the disease. Five times within a couple of months I have found supposed extirpations of Cancer to be demonstrably incomplete. I am far from wishing to convey the impression that the operators were themselves in fault in these cases. That which is to be gathered from the fact rather is that, when practised Surgeons meet with such difficulty in eradicating cancerous tumours, the subsequent recurrence is not due to any abstract quality of the disease.

The following cases are arranged with a view to illustrating the several methods of inadequate operating, and of throwing light upon one another. In some the tumour only was taken away; in some a greater or less proportion of the mamma was removed with it; and in others, while the organ was extirpated, diseased tissues near it were left.

**CASE 1.**—*Recurrent Cancer near two several wounds on the surface of the mamma.*—I admitted into the Middlesex

Fig. 1.<sup>1</sup>



The central striped parts indicate the scars succeeding operations for the removal of separate cancerous tumours of the breast. The crossed lines around them show masses of Cancer which began to grow, in the one place before the healing of the wound, and in the other just after it.

- Hospital a tall woman, of about 38 years of age, having a recent scar and an unhealed wound on the surface of her right breast. The scar was already the seat of cancerous thickening, small, flat, but unequivocal. The skin of her chest was coarse, being covered with the white, yellow, raised or pitted little scars of a phthisical patient. She was quite free from pain in the chest; yet her respirations were forty in the minute, and Dr. Henry Thompson distinguished dullness at the apices of both her lungs.

She was married and had a family. Two superficial tumours formed in the right breast a year and a half ago; and nine weeks ago they were cut separately out of the breast.

It appeared certain from this immediate recurrence of the disease in and around the scar, and there only, that the tumours had been removed without the intervening and out-lying fragments, and that nothing short of a complete extirpation of the breast could have comprehended them all. But, notwithstanding the possibility of still accomplishing that operation, I was led by her general condition and especially by the state of her breathing to dissuade her from submitting to it.

She went home, but during her short stay in the Hospital Cancer recurred in the other scar.

*CASE 2.—Recurrent Cancer in a large portion of the right mamma after an operation limited to the centre of the organ.—*This patient looked healthy, but she presented a rather large amount of local disease. The central parts of her right breast having been removed, together with a cancerous tumour they contained, she had recurrent Cancer in the residue of the breast, with disease of the glands in the right axilla, as well as above the right clavicle.

The scar was in a pit surrounded by large tuberos elevations, making altogether a mass greater by half than the other healthy mamma, but crater-like instead of highest in the middle. One of the great lobes was softening, and the thin skin covering it was of a dark purple colour. Except at this

softened lobule and the scar, there was no adhesion of the tumour to the skin, and it could be moved with some freedom

Fig. 2.<sup>1</sup>



over the pectoral muscle. Apart from the principal mass, and beneath the skin above and within it, could be felt a movable hard nodule, larger than a cherry stone. Such a nodule, if found by itself at a time long subsequent to the extirpation of the breast, might be supposed to be an independent and spontaneous disease; but its growth simultaneously with, though somewhat apart from, the chief recurrent disease demonstrates a connection between them, which is of importance in judging of the source of a similar nodule when observed alone. See Case 10.

In Cases 1 and 2, the disease recurred in the textures which had been divided in the operation, and spread in every direction from beneath the scar. The return in the second case was not indeed so rapid as in the first patient, several

The oblique stripes in the middle of the disease indicate the position of the scar, around and beneath which are lobes of Cancer, somewhat inaccurately represented. Scored circles in the right armpit and the neck correspond to cancerous glands. A little above and internal to the inner and upper lobe is one small subcutaneous isolated cancerous nodule.

months having elapsed before any renewal of the growth was recognised. In a couple of years, however, the remnant of the mamma was all involved in it, as well as the glands both below and above the clavicle; whilst in its increase towards the sternum it had led to the formation of an isolated cancerous nodule in the midst of healthy subcutaneous tissues. From later cases it is probable that a lymphatic was the unseen channel of communication between the primary disease and this tumour near the sternum, as was certainly the fact in its extension to the glands.

Though equally disastrous, the effects of this mode of operating are not always alike; and another case of the same kind may be quoted with advantage in this series, as it illustrates a different progress of the disease after an operation purposely limited to the mere excision of the tumour.

*CASE 3.—Recurrence of Cancer, chiefly in the axilla, after a limited operation for scirrhus of the mamma.—*

A scirrhus tumour was cut out of the right breast of a lady by a horizontal incision above and outside the nipple. Fifteen weeks afterwards she came to England, and I saw her with my colleague, Mr. Hulke. The scar was a model of evenness and neatness, and the form of the breast was scarcely altered. There was also no distinct hardness or tumour beneath the scar, though there may have been some thickening, and the remainder of the mamma was tender. Some of the glands, however, having lately enlarged, formed a cluster of firm tumours in the right axilla. The highest limit of the disease in the armpit was a little uncertain, but there was no Cancer above the clavicle.

It was recommended that the axilla should be opened, and that, if the whole of the diseased glands could be taken away, the breast also should be removed; but that the latter operation should not be proceeded with, if diseased glands were to be left behind the pectoral muscle.

It being found impossible to clear away all the glands, Mr. Hulke forebore from amputating the breast. He has since

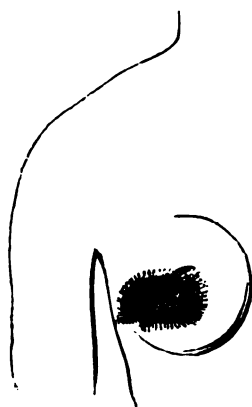
informed me that, though the disease in the mamma proved to be cancerous, it did not afterwards much increase; in the axilla, however, it continued to grow and to fill that cavity, until in less than a year afterwards she died.

Though neither from beneath the areola nor from the surface of the mamma is Cancer extirpated by the removal of the tumour only, it may still be questioned whether it is needful to sacrifice the whole breast when the tumour implicates but one edge of it. The disease then has such an appearance of isolation, and the remainder of the breast is so perfectly soft and healthy, that Surgeons cannot but be tempted to remove the diseased edge alone, or at most that segment of the organ in which the tumour is seated. Especially if the disease be recent, is there danger that this method of operating may be adopted. Indeed it cannot be denied that such operations are sometimes followed by an interval of health which, in contrast with the earlier recurrence of older and more diffused Cancers, the Surgeon may deem satisfactory. But that mode of judging the result is surely fallacious. The question should rather be thus stated: if persons so operated on survive a given number of years and then suffer a local recurrence of their disease, how much longer exemption would have followed the removal of the whole breast? Surgeons who thus operate do less for their patients than the case allows, and miss the best hope we know of permanent cures. It is just in these cases of recent and limited tumours, that the total extirpation of the mamma obtains its fullest reward. Whereas, if the removal be incomplete, it is in the breast which should have been taken away, or in a structure closely connected with it, or most commonly in the very site of the operation, that a fresh outbreak of disease proves the error which has been committed. Were this method of operating right, there should happen cases in which Cancer arose in a distant organ, while a portion of a mamma from which a primary Cancer had been removed remained exempt from the disease. I have not met with such cases.

CASE 4.—*Recurrent mammary Cancer in the site of a former operation limited to the outer portion of the breast.*—A patient, æt. 46, rather stout and pale, was brought to me, from the outermost edge of whose right breast a cancerous tumour had been removed fifteen months before, and the disease had returned.

The margins of the breast appear healthy above, within, and below. The central and outer parts are hard, broadly depressed, and covered with adherent pitted skin, in which the

Fig. 3.<sup>1</sup>



nipple lies, shrunken and drawn in. Immediately above the depressed surface the skin is soft and thin, but it is thickened chiefly, if not only, by œdema, over the whole of the lower part of the breast. The broad mass of Cancer occupies the entire depth of the breast, and adheres to the muscle beneath. The scar of the operation is linear, and crosses the outer part of the disease obliquely from without, upward, and inward; two inches at either end of it are perfectly soft, healthy, and unadherent to the textures beneath it; the middle part, though still discernible, is mixed up

<sup>1</sup> The diagram represents a mass of solid Cancer, involving the central and outer parts of the right breast, together with the nipple and the middle of a long oblique scar.

with the diseased mass. Some of the lower axillary glands are large and hard.

This disease becoming painful, and being apparently capable of removal, I repeated the operation. The soft healthy upper skin I cut near the diseased; the lower, which was œdematous, at about an inch and a half from the diseased. The scar was divided in the two parts where it crossed the long oval incision. I turned the flaps off from the breast, which I detached first at its edge, and then removed in one mass with an adherent piece of the muscle and with the diseased axillary glands. Though nearly certain that the skin at the lower part of the incision was œdematous only and not cancerous, I touched its edge with the solid chloride of zinc. The wound was made in large lobules of fat, and before closing it, I sponged it freely with Zinc. Chlor., grs. 40 to an ounce of water.

The wound healed in thirteen weeks, and the patient went to Brighton, but with doubtful thickening near the scar.

Four months after the operation I saw her again, and found her in the fullest health. The disease had resumed its growth, and formed a low nodular hard tumour on the side of the chest, in connection with the axillary end of the second scar, and with thickened skin just below it. Not being freely movable, it was probably also attached to the subjacent muscle. It was three inches broad at the scar, from which it extended three inches upwards into the axilla, and downwards two inches. The integument above the scar was soft and healthy, but that below it was tense, partly from œdema and partly from complete contraction of the wide wound to a linear cicatrix.

With so great an improvement in her general condition, it was impossible to attribute the recurrence to an increase of constitutional disease. There was now none which could be discerned amid healthy functions, healthy look, and healthy feeling; yet the local tumour had returned with a vigour greater than after the first operation. Neither was the recurrence diffused, either in the body, or over the mammary region, or even throughout either scar; but it was limited to



just those portions of skin and muscle and abundant fat which had most nearly adjoined the disease, and being suspected had been in part taken away. The result shows an error in my operation on the side of defect, and also, by a strictly local recurrence, a local disease.

**CASE 5.**—*Cancer recurrent in the breast beneath the scar of an operation.*—A very stout woman, æt. 45, consulted Mr. John Scott, and was sent by him to me. At the inner and upper margin of her left breast was a circumferential scar, of four or five inches in length, with marks of needle punctures beside it. The scar and skin were soft, but the former was attached at one part to a tumour in the substance of the breast. The tumour was as large as a goose's egg, uneven, hard, covered with breast tissue and adherent to it. The mamma being very large, the tumour did not reach the nipple, which was not retracted. In each axilla was an indistinct nodule, feeling like an enlarged gland, and that on the right side was larger than that on the side of the diseased breast. There was no glandular disease in the neck.

The patient had been a healthy woman: she had had five children, the youngest five years ago. All her labours were difficult, and the last child was perforated. The tumour had been removed two years, but eight months ago it reappeared.

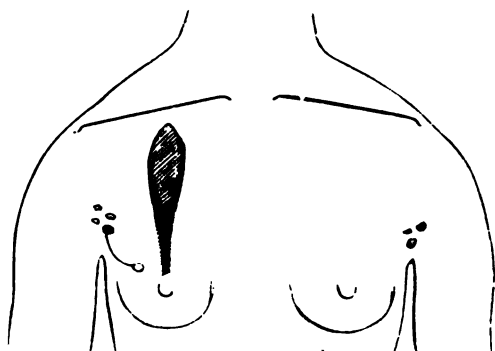
I advised removal without delay, and she returned to the country to have the operation done at home.

The lesson furnished by this case is the same as that of those which precede it. Recurrence was neither constitutional nor organic, nor regional, but that of an incompletely extirpated tumour.

**CASE 6.**—*Cancer, recurrent after a partial removal of the breast.*—A thin and pale sempstress, æt. 54, had a long vertical scar, extending almost from the clavicle to the right nipple. The highest part of it was about an inch in breadth, soft and healthy; the lowest part was narrow and somewhat sunken in the breast. That organ itself was flaccid and thin,

and it appeared to be healthy; but on its surface, an inch outside the lower extremity of the scar, was a firm cancerous

Fig. 4.<sup>1</sup>



tumour, of the size of the end of the little finger, movable, subcutaneous, not distinctly attached to the breast or to the scar. Thickened lymphatics passed outwards from it under the pectoral to some enlarged and firm axillary glands. There was also a slighter enlargement of glands in the left armpit; the left mamma was healthy.

A tumour at the upper part of the right mamma attracted the patient's notice several years ago. It was at that time of the size of a walnut, but as it caused no pain and did not distract her from her hard work, she paid it no further attention, and only afterwards remembered it. When, however, seventeen months ago, it had grown upwards in the form and size of a pear, and nearly reached the collar-bone, it was removed, without the breast.

I first saw the patient after the tumour and adjoining tissues

<sup>1</sup> A vertical scar, represented by oblique lines, extends from the upper part of the right breast nearly to the clavicle. The original tumour began in the mamma, and the recurrent tumour an inch on the outside of it. The line drawn from the latter tumour to cancerous glands in the axilla is intended to represent thickened absorbents, which may be easily felt in the patient. The glands in the left armpit are likewise somewhat enlarged.

- had been freely injected with acetic acid, and found the lowest part of the scar thickened, and apparently connected by tumid breast tissue with the tumour. Mr. De Morgan, under whose care the patient had come, informed me that the lowest part of the scar was not dense before treatment. Without this condition, however, the connection of the first tumour and the recurrent disease was distinct. Both sprang from the upper edge of the breast, the former growing from it upwards, and the latter being an invasion of the lymphatics by morbid matter originating in that same spot alone. No operation could have been effectual in such a case, by which the least morbid portion of the breast itself was left behind.

In the next cases there had been an intention of removing the whole breast, but some small portion of it was left behind. This may perhaps not unfrequently occur, and the remnant of the mammary substance may gradually wither and be absorbed. But I have had no opportunity of ascertaining the fact, and whether such involution be or be not usual after incomplete removals of the breast, the following cases show that event to be not invariable. Now if, when a portion of the organ has been left, the renewed disease begin in distant or in various parts of the mammary region, its reappearance may be held to be uninfluenced by the method of operating; whilst an outbreak of Cancer in the remnant of the mamma, and its dispersion from thence only, prove at once the exclusive connection of that part with the foregoing disease, and the correctness of the theory of its local origin. Such a fact indicates clearly the propriety of a complete removal of the organ in which the disease arose, and establishes the theoretical inference drawn from previous cases in which a larger portion of it was designedly left.

CASE 7.—*Cancer, probably recurrent, in a small internal portion of the mamma left in a previous operation.*—A patient came under my care in 1866, from whom the right breast had been removed a couple of years previously. I found a vertical soft broad scar on the right pectoral

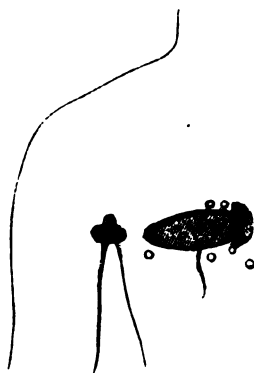
muscle, and towards the sternum a subcutaneous firm prominent movable tumour of twice the size of an almond. From its position over the costal cartilages, on a horizontal level with the middle of the scar, and distant from it about half its length, or the radius of the breast, there could be no doubt that the tumour was a portion of the inner edge of the breast, which had become thickened. There was no other disease on the surface of the chest or in the axilla, but the patient was uneasy about the tumour, and complained of various pains about the right side of the chest. She was out of health, and looked phthisical, and whilst under my observation she had a bilious attack and some painful hæmorrhoids.

As the tumour itself, though dense, was not distinctly hard, nor was it the seat of pain or tenderness, I could not be certain that it was the cause of her ailments. After watching it therefore for two or three months, and finding that it did not increase, I let her go away. Nevertheless, it now appears to me by no means certain that her uneasy feelings may not have been due to the existence of Cancer in the thickened remnant of the mamma, if not also to its extension through the intercostal absorbents to glands within the chest.

*CASE 8.—Recurrent Cancer in a remnant of the mamma, infecting the subcutaneous lymphatics and the axillary glands, but not the scar.*—A lady, aged about 40, was brought to me two years after the removal of her right breast. The operation had been a very thorough one, and the scar was large. It was thin, pliant, and movable on the subjacent textures. Beneath the skin at its inner edge was a three-cornered hillock, shaped like a cashew nut, precipitous outward, bevelled towards the sternum, and about an inch and a half in its vertical measurement. Its outer surface was firm, and the slightly thickened skin covering it showed enlarged pores when lifted in a fold. The mass appeared to be the innermost edge of the mamma partially infiltrated with Cancer. There was no disease in or beneath the scar, but along the

upper and lower borders were several cancerous nodules, of the size and slipperiness, and almost of the hardness of peas.

Fig. 5.<sup>1</sup>



The skin covering these nodules had no adhesion to them. Behind the edge of the pectoral muscle lay four enlarged and hardened glands, closely adjacent but not adherent to one another. A cord of lymphatic vessels led upward from the highest of them, and was lost behind the pectoral. No other gland could be felt in the armpit, and the space above the clavicle was healthy. There was no disease in the left mamma. The recurrence of these growths had not much attracted the notice of the patient, who, however, had spoken of pains for about six months.

There could not be a more definite illustration of the local characters of Cancer. By an extensive operation the original tumour, with the breast and a broad covering of

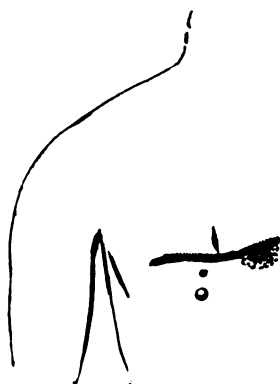
<sup>1</sup> The oblique stripes and the line depending from them represent the scars remaining after the removal of the right mamma. Internal to the scar is a fragment of the mamma, cancerous; parallel with the scar, both above and below it, are subcutaneous cancerous nodules, believed to be connected with lymphatics, by which also portions of the disease have been conveyed from the principal tumour at the inner end of the scar to the glands, represented by scored circles, in the armpit.

skin and superficial absorbents, had been removed, and there was no recurrence in the scar. A small part of the inner edge of the mamma having, however, been left, the disease had continued to grow in it. This portion now constituted a primary tumour, and from it, as a new centre, the growth disseminated itself in the usual way, passing by the lymphatics to the nearest glands in the axilla. But, there being no lymphatic canals in the scar, which was in the direct line to the armpit, the morbid matters had taken a circuitous course above and below it, and had formed small subcutaneous nodules, such as we are accustomed to find by dissection in the track of lymphatic vessels. Full and probably thickened absorbents in the axilla showed that the disease was already passing in the same manner into yet higher sub-clavicular glands. Moreover, from the existence of one or two nodules lower down on the chest than the area of the operation, and out of the line of lymphatics between the principal tumour and the scar, there were indications that the disease was ready to extend in other directions than towards the armpit.

In the next series of cases the entire breast appears to have been removed. It is of course difficult to secure absolute certainty of the removal of all the mammary structures, as the precise limits of the breast are ill-defined, and vary much in different persons. But there is every probability in the following cases that the operation, so far as the breast is concerned, was complete.

*CASE 9.—Recurrent Cancer in skin and cicatrix, infecting the subordinate lymphatics and glands.* — In December, 1865, Mr. Square, of Plymouth, removed the right breast of a slender woman, aged about 44, and subsequently by a second operation took away a small fragment of apparently recurrent disease below the innermost end of the horizontal scar. On a further return of the disease he recommended the patient, who had come to reside near London, to apply to me, and I saw her in May, 1866.

I found that the right breast had been completely removed, and in its place was a thin and nearly horizontal scar, movable over the right pectoral muscle, and forked at its inner end. About the lower of these branches of the scar there

Fig. 6.<sup>1</sup>

was some shooting pain, and a little enlargement of the pores of the skin. Low in the right axilla was a globular, firm, tender, slippery gland, as large as the end of the little finger. Three weeks afterwards there were two enlarged glands in this armpit, and I advised her to have them removed. After a further delay of a month this was done. But before the autumn she returned to me with a hard pea-shaped nodule fixed in each of the inner branches of the scar, and with two similar but perfectly movable subcutaneous tumours a little below the middle of it. The axilla was now healthy.

The explanation of this occurrence appears to be that some of the original disease remaining in the skin at the inner end

<sup>1</sup> In the place of the right mamma is a horizontal scar, forked at its inner end. The dots represent slightly thickened skin; the circles show nodules of Cancer, the inner two being cutaneous, the lower two subcutaneous; the line in the armpit indicates the scar left after the removal of some cancerous glands.

of the wound multiplied at first in that structure, and afterwards entering a lymphatic, began to grow on reaching the axillary glands. The removal of these glands checked the ascent of the disease to higher parts of the axilla, but did not lessen the supply of morbid matter from the sternal end of the scar. As this increased, it assumed the form of little tumours, and continued at the same time to fill the lymphatic channels connected with it. But the axillary extremities of these vessels having been closed by the last operation, the morbid matters accumulated and formed tumours in their course. The removal of lymphatic glands, and their complete destruction by Cancer, thus produce equivalent effects. They dam up the current in the afferent lymphatics and cause its accumulation in tumours, so long as the primary texture continues to furnish cancerous matter. Accordingly it now appears to me that my operation of removing the glands alone was not an adequate operation. More importance has since attached to the merely thickened skin; and in the subsequent progress of the case it has been shown that that small factor of morbid material, though attracting little attention at first, should likewise have been removed.

Into each of the four nodules I injected acetic acid in the manner proposed by Dr. Broadbent, and found them in three or six weeks either much reduced in size or no longer perceptible. But in the course of a couple of months each tumour had grown afresh, those which had disappeared arising first as a soft swelling, and then regaining their former hardness and globular shape. After this event had repeatedly occurred, I injected each nodule somewhat freely with undiluted acetic acid. The outer two nodules, which were entirely subcutaneous, subsided; the inner two, being implanted in tough scar and thickened skin, sloughed. The further effect of this destruction of the residual disease, on which depended the infection of the lymphatics and glands, is not yet manifest.

*CASE 10.—Recurrent Cancer near the inner and the uppermost extremities of the scar, after an extensive operation*



*for the removal of the right breast and its axillary glands.—*

In 1859 I removed the right breast of a patient, who had fifteen months previously discovered in it a cancerous tumour of the size of a nut. The operation was an extensive one, and having taken away the axillary glands with much tissue, I put a drainage tube through the back of the armpit. The patient recovered health and was perfectly well for four years, when she began to have pain along the nerves of the right arm. In the following two years that arm became œdematous and weak, and she carried it in a sling. On her applying to me again six years after the operation the arm was much swollen and weak, but the pain in it had diminished. The scar was wide and adherent to the chest, but both it and the adjoining structures were perfectly healthy, and I could discern no tumour whatever in the axilla. Neither was there any swelling above the clavicle, but as she could not turn her neck freely, I examined it closely and found that, with

Fig. 7.<sup>1</sup>



<sup>1</sup> The scar, much too small and regular in form, as well as deficient towards the axilla, is indicated by an obliquely striped line. A cluster of dots in the neck points out a small spot at which the skin is dimpled over a small, fixed, cancerous mass about the brachial plexus. This was discovered six years after the operation, though it may have been growing from the fourth year. Above and within the inner end of the scar, as in Case 2, a small, isolated, subcutaneous nodule, formed seven years after operation.

the head inclined to the left, the right scaleni dragged upon a tough spot near the right brachial plexus, over which the skin, without being thickened, was a little adherent. The dense substance in this spot was not large enough to form a swelling.

After another year the œdema had under treatment almost subsided, and the arm was without pain; but it was so weakened that it might be said to be partially paralysed. Sensation was unimpaired, and the fingers could be moved, but there was no power over the shoulder-joint, elbow, or wrist, and the limb could not be raised into the sling without the help of the other hand. No increase had taken place in the hard thickening beside the lower cervical vertebræ, and the skin was even a little loosened from it, but the scaleni were more drawn. Examining the chest again, I now found one cancerous nodule beneath the skin over the sternal end of the right second intercostal space. It was a little larger than a cherry-stone, and was situated so far from the scar as to be out of the area of the wound made in the operation. It was moreover quite isolated, no remnant of the breast being anywhere perceptible. In December, 1866, I injected the nodule and the dense substance at the root of the neck with diluted acetic acid, and found both of them diminish. The skin became looser in the neck, and that over the shrunken nodule on the chest sunk in and showed a visible dimple.

This case is perhaps an extreme instance of the uncertainty of the distinction between reawakening of a long dormant residue of living Cancer, and a fresh outbreak in the region, as distinguished from the organ, first attacked. The first recurrent Cancer, which was in the neck, appeared, from its position, to be unconnected with the original disease. For whilst in all the former cases recurrence was first observed about the scar, and the subsequent disease involved the axillary or the supra-clavicular glands, in this case it arose amongst the nerves, and was first discovered in relation with the scaleni, the brachial plexus, and the transverse process of the lowest cervical vertebra. The form of the growth when first observed did not indicate an origin in a gland, and

it was not till a year afterwards that I found the nodule above the scar. These difficulties are, however, only apparent. The form of recurrence could hardly have been usual after so extensive an operation, and in a case in which the rate of the subsequent growth of the disease appeared to lengthen. Had the first renewal taken place over the pectoral, the breadth and adhesion of the scar would have obstructed its course by the lymphatics towards the axilla, and diverted it over the clavicle to the glands in the neck. There would thus have been no advance of the disease to the armpit, even had any glands been left there, which could have received it. The only other glands which could have been infected by this superficial recurrent growth, namely, those above the right clavicle and in the left armpit, were healthy. It appears clear, therefore, that the cancerous growth about the brachial plexus could not have arisen in continuity with the small subcutaneous tumour near the sternum; and the course of the symptoms associates it with some fragment, which was left among the nerves in the highest part of the axilla, and which began to grow at the time of the first pain in the arm.

Though the disease first noticed was in the neck, and the nodule over the chest was not found till a year afterwards, it is difficult, though it might not be unreasonable, to regard the latter as the primary. For, when glandular disease is the first to attract attention, it is not uncommonly excited by a primary Cancer of so small a size as to be discernible only at a later period of its growth. Inasmuch, however, as the cervical disease does not appear to have been glandular, there is no need to suppose any such exceptional explanation, but to connect it with an axillary portion of the original disease. I add another of my cases which seems to bear out this opinion.

CASE 11.—*Probably recurrent Cancer in the axilla six years after an extensive operation.*—Emily N—, æt. 45, came under my care in 1855, having had tenderness in the left breast for twelve months, and more recently tenderness and shooting

pains in the right breast and whole right side, with headache and other symptoms, which appeared to arise from the excessive use of tea, and which subsided under treatment.

The breasts were very large. The left was natural, except at the upper part of its axillary border. At this part there was a slight pit, the skin was thickened and adherent, and the mammary tissue was found on superficial examination to be nodular and a little hard, but no distinct tumour could be felt. At the corresponding spot in the other breast, where the axilla and breast met at the edge of the pectoral muscle, was a similar pit, without thickening of the skin. The left nipple was not retracted; the left axilla appeared to be healthy; the left external jugular vein was more prominent and larger than the right.

November, 1855.—I made an incision into the thickened part of the left breast, and, finding there a small but distinctly scirrhus tumour, I removed the whole organ. The wound was large, but few vessels bled.

April, 1856.—She complains of her right breast, but there is no growth in it.

June, 1858.—There is no return of the disease; the cicatrix is sound, and her health is good. There is no tumour in the right breast, to which, however, her attention seems much directed. She has a few sebaceous tumours on the scalp.

June, 1863.—She has applied to be a Cancer out-patient, and has been kindly sent by Mr. Nunn to me. She was stout and apparently in good health, but her breath was short, and she had severe intermittent tingling or scalding pains in the left fingers, or in parts of the left arm which she indicated with great precision. These had been first noticed in November, 1861, but they had lately increased. She described them as like the burning of molten lead or boiling water, and her only relief, until they went off, was obtained by soaking the limb in hot water or flannels. The scar was healthy and perfectly loose. The left side of the neck, the whole scapular region, the pectoral and the arm, were oedematous, moderately but plainly so. There was no trace of disease in any axillary gland.

November, 1863.—The pain down the arm continues

intense; the movements of the shoulder are restricted, but the arm is little swelled. The pectoral muscle is raised, and the hollow of the axilla is deeper than before. At its highest part is a small ulcerated surface, not distinctly cancerous. Two or three glands above the clavicle are enlarged and hard.

Her sufferings were mitigated by the application of morphia to a blistered surface on the left side of the neck, and by subcutaneous injections of the same salt. They continued, however, to be very severe, and towards the end of her life her breathing became peculiar and difficult, apparently from some affection of the left phrenic nerve by the disease in her neck. This, however, could not be verified, as, on her death, which took place in the spring of 1864, permission to examine her body could not be obtained.

Notwithstanding the long period of six years which elapsed between the operation and a renewal of symptoms in this case, I cannot doubt that I had incompletely extirpated the disease. Some cancerous invasion of parts behind the left clavicle, and therefore beyond reach, must have already occurred when I removed the breast. Though much protracted in its rate of growing by the loss of the primary tumour, the disease proceeded in one direction just as if it had remained in continuity with the mammary Cancer, involving the subordinate glands only and the adjoining nerves. In every other direction it ceased.

A longer interval than any yet given may elapse between the removal of a cancerous tumour and the local return of the disease. So late indeed may be the recurrence that it is not surprising to find it regarded as the outbreak of a new and independent disease. Yet in all but its delay the recurrence is precisely similar to that which has been noted in previous cases.

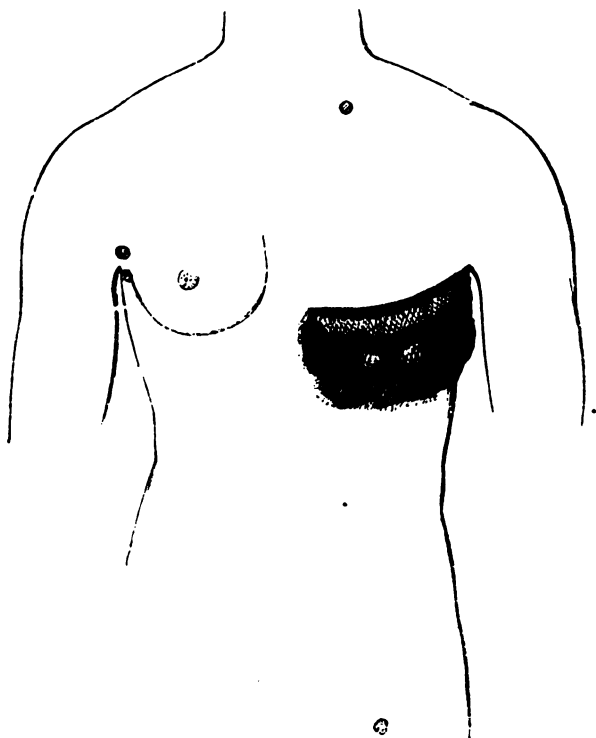
*CASE 12.—Cancer, recurrent at the border of the scar more than nine years after the removal of a recent scirrhus of the breast. Further exemption from the disease, already known to have lasted for seven years, after a second operation.—*

Sarah H—, æt. 47, was one of a family of six sisters, five of whom and their mother suffered from Cancer of the left breast. The tumour in the patient Sarah H— was removed by Mr. Arnott in December, 1846, four weeks after it was discovered. It was about the size of the last joint of the thumb, and situated around the nipple, which, however, was not in the least retracted; and it was an example of genuine stony Cancer. From the time of the operation she enjoyed good health, until the beginning of 1856, when she perceived a return of the disease in the cicatrix. In April of that year there was at the axillary border of the cicatrix a hard tumour of about the size of a small pigeon's egg, adherent to the integument, though free over the surface of the chest. In the axilla and above the clavicle were some enlarged but not indurated glands. This lump, together with four axillary glands, was extirpated by Mr. Shaw, and both to the naked eye and under the microscope the whole was found to be made up of well-marked somewhat soft scirrhus.

It is hard to include among inadequate operations one which was for so many years effectual. Yet, if the recurrence were due to delayed growth of some fragment of unhealthy tissue which was left at the time when the principal mass was removed, it must be so included. That this was really the case appears proved by the position of the renewed disease. Not in a distant part, not in an internal organ, not even in the remaining breast, but in close proximity to the scar, and on that side of it which was in the direction of natural dispersion of the first Cancer towards the axilla, this recurrence connects the second with the primary tumour. The remarkable continuance of health after the first operation moreover dissociates the disease from any separate accumulating constitutional ailment; and it further shows the dependence of internal Cancer, and even of the infection of the second breast, upon a certain amount or mass of the local tumour, which this early and admirable operation reduced. In the absence of growing material, diffusible from the primary Cancer, the whole of the remainder of the body continued healthy.

*CASE 13.—Recurrent Cancer below a transverse scar, after the removal of the left mamma and its axillary glands. Long abeyance of the disease above the scar, especially in a*

Fig. 8.<sup>1</sup>



<sup>1</sup> The diagram represents a large mass of Cancer abruptly limited at its upper part by a scored transverse line, which indicates the position of the scar. A striped circle in the neck marks a cancerous gland, diseased at the time of the operation, and unaltered afterwards (notwithstanding the exuberance of the growth below the scar) until in a later stage of the case the disease broke through the scar and flooded the healthy textures on the wall of the chest above it, as well as the glands in the neck. Striped circles in the right armpit and the left groin show the position of glands to which disease was conveyed by collateral lymphatics from the recurrent Cancer on the left side of the chest.

*cervical gland, which was enlarged at the time of the operation, and which increased so soon as the disease below had destroyed the scar.*—The next diagram represents a case in which I removed a large cancerous left breast with the axillary glands, deliberately leaving a solitary, enlarged, globular and movable gland behind the sterno-mastoid muscle of the same side. Though it was my intention to remove the entire mamma, and it is my belief that I did so, yet the disease speedily returned below the scar. The course of the recurrent disease, and that in the cervical gland, which were thus separated by the scar, presented a singular and instructive contrast, the conditions of which were ordinary, and intelligible without reference to the constitution.

The recurrent mammary disease increased below the scar, and in the course of three or four months became a large mass of great prominence and breadth, abruptly separated by the scar from the healthy structures. Immediately above the scar the integument was level, soft, pale, thin, movable on the pectoral, and indeed perfectly healthy. Immediately below the scar the disease rose in a precipitous swelling, distended with œdema and venous blood and covered with turgid skin. The gland behind the sterno-mastoid remained no larger than it had been before the operation. Though in the same constitution, and nourished by the same blood as the disease which was so active below the scar, it continued absolutely quiescent. After some weeks more, two glands became cancerous in the right axilla, though no tumours could be felt in the right mamma; and it appeared that the great mass of disease, which now covered most of the left side of the chest from the scar downward, and was approaching the mesial line, had infected the glands of the right armpit through collateral lymphatic circulation. Meanwhile the gland behind the left sterno-mastoid remained unaltered. At length, however, the disease passed through the scar, and began to form small cancerous nodules in the healthy skin above it. The effect of this upon the glands above the clavicle was immediate. In three days they were perceptibly increased, and that dis-



eased one behind the left sterno-mastoid, so long unaltered, now distinctly enlarged. Three weeks later it had nearly doubled in size, and it was united in a dumb-bell shape with an adjoining gland which, though previously imperceptible, had grown to be nearly equal to it. From that time disease spread rapidly both above and below the clavicle. The scar became obliterated in the growing and ulcerated mass, and when the patient died, the superficial collateral lymphatic circulation had conveyed some of the morbid material to one of the glands in the left groin.

The inference from this striking case plainly is, that the extension of the disease was that of a local ailment only. It spread by continuity of the morbid material: where tough scar interposed, its progress was arrested; existing disease, if not continuous with the principal local mass, was in abeyance, no assumed ailment of blood stirring it to the least activity; and there was a total exemption of other parts of the body from the disease, except such as were in communication with it by lymphatics. To search in the constitution for the source and control of an ailment so definitely ruled by local conditions appears superfluous. This, however, may be added in explanation of the peculiarities of the case. The cancerous cell elements formed an unusually small proportion of the substance of the disease. They were mixed with an abundance of a thick liquid, having some resemblance to the cream of milk; and by this, whilst the transfer of the cells from place to place was facilitated, their destructive action on the natural textures and their multiplication when transplanted were probably hindered. This curious association with a growth of Cancer appears to have been a functional disturbance, and to have been essentially an excessive secretion of milk. From another case which has occurred to me there appears reason to think that the tendency to it may be taken up and continued by textures adjoining the breast, though the product of the undue activity of those textures is of course not milk, but such a liquid as their construction allows them to exude.

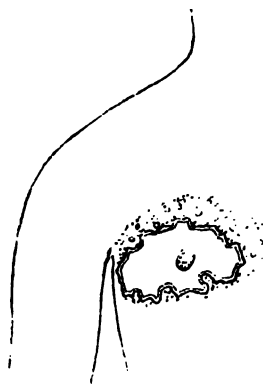
Seeing that the disease was so distinctly limited, it may be

asked whether a more extensive operation might not have availed to eradicate it. My own impression is that this would have been improper. The operation which was performed was a large one, and during the time when the local recurrence was limited to a few small tumours, the general condition of the patient did not admit of the operation being repeated. The case illustrates none the less the consequences of a partial removal of the disease, and a continuity of the morbid growth over a very wide area of the body.

The disastrous result of leaving the skin and nipple when the breast is removed is illustrated by the next case.

CASE 14.—*Recurrent Cancer after an operation in which the breast was removed, but the skin covering it and the nipple were retained.*—A patient came to me in much suffering from recurrent Cancer and dyspnœa. In the place of the right breast was a shallow hard-edged ulceration, measuring horizontally  $4\frac{1}{2}$  inches by  $2\frac{1}{2}$  inches vertically, and surrounded

Fig. 9.<sup>1</sup>



<sup>1</sup> In the centre of the diagram is the cancerous nipple, surrounded by a large ulcer, which has destroyed the remainder of the flaps of skin, and beyond the sinuous edges of which the thickened integument, with pores enlarged and beset with cancerous tubercles, is indicated by dots and small circles.

by dense adherent skin, which was thickly studded, especially above, with cancerous tubercles. Isolated in the midst of the almost level ulcer was the nipple, also diseased. The ulcer was in an irritable state, and there was bright inflammatory redness of the integument down to the lower margin of the chest.

The patient was an Irish washerwoman, who appeared to be 30 years of age, but supposed she might be 40. Her history was that the right breast had been removed eight months previously for Cancer of ten months' standing, and by an operation in which the nipple was not removed, but the skin covering the breast was raised in a flap and then laid down again upon the pectoral muscle. The wound healed quickly, but not completely; it soon reopened; all the flap ulcerated except the nipple; and on her applying at the Middlesex Hospital every structure in the area of the wound made in the operation, and for some distance around, was cancerous.

There is one mode of recurrence of Cancer almost exclusively peculiar to persons having much subcutaneous adipose substance. It has been already made known by my colleague, Mr. De Morgan, in his valuable essay on the use of solutions of chloride of zinc after operations, and in recent wounds. Nodules of Cancer spring up in these patients in close proximity to the scar and in its whole length, or (which is an equivalent fact) in the whole area of the wound made in the operation. The recurrence is sometimes so speedy, and the appearances on repeating the operation are so decisive, as to leave no question that each nodule is the product of a residual fragment of disease, which was concealed at the time of the operation in exuberant fat. Though the texture holding the seed of the too certain crop is different, the occurrence is identical with that in the preceding case, in which a multitude of germs were folded back upon the wound with the flap of skin and nipple. It is not every amputation of a mamma beset with much fat, which is followed by so diffuse a growth of Cancer; and there is reason to think that great care in taking away this superfluous substance might be attended with a result as satisfactory as that in Case 10.

Though the foregoing cases may not illustrate all the phases of recurrent Cancer of the breast, they appear to bear with a decisive uniformity on the questions respecting the nature of the disease. It is possible that there may be methods of recurrence which would favour other conclusions than those I am about to draw : but as my cases have not been selected to prove an opinion already formed, I am satisfied that they represent the usual occurrences. They are also in accordance with examples of Cancer, which have been demonstrated to recur in the same manner in other organs. The following are the conclusions to which my cases lead.

Local recurrence of Cancer after operations is due to the continuous growth of fragments of the principal tumour.

Such recurrence may take place also in a residual part of the organ, respecting which it cannot be asserted that it was cancerous at the time of the operation.

Such recurrence may further happen in a structure adjoining a completely extirpated breast, and on a comparison of cases may be held to be produced by disseminated fragments of the original tumour.

The recurrent Cancer is subject as well as due to local conditions, and especially adapts itself to the distribution of the absorbents. Its continuity with the first tumour may be traceable over half the chest, the pleura, and the glands from the neck to the loin or the inguinal region, and possibly also to the liver.

After the removal of a portion of the breast, the recurrent Cancer does not involve the remainder of the organ indiscriminately, but commences in that part of it which immediately adjoins the scar.

The progress of recurrent Cancer after a partial removal of the breast is not exclusively organic, and does not even show a preference for that organ, but rather is centrifugal from the scar ; and, when free in both directions, it tends toward the axilla earlier than to the residue of the breast.

Cancer once established in either mamma, its primacy is thenceforth supreme. When one breast has been wholly or partially removed, recurrent Cancer does not spring up as

a new disease in the opposite breast, but on the same side as the original tumour. It may, nevertheless, be possible that, a mammary Cancer having been wholly extirpated, Cancer may reappear in another part of the body, which other part may be the remaining breast. I have not met with such a case.<sup>1</sup>

It is not sufficient to remove the tumour, or any portion only of the breast in which it is situated; mammary Cancer requires the careful extirpation of the entire organ.

The situation in which the operation is most likely to be incomplete is at the edge of the mamma next the sternum.

When any texture adjoining the breast is involved in or even approached by the disease, that texture should be removed with the breast. This observation relates especially to skin, to lymphatics, to much fat, and to pectoral muscle. The attempt to save skin which is in any degree unsound is of all errors perhaps the most pernicious, and whenever its condition is doubtful, that texture should be freely removed. A broad scar, and the stretching and compression due to its subsequent contraction, appear to be especially satisfactory.

In the performance of the operation it is desirable to avoid, not only cutting into the tumour, but also seeing it. No actually morbid texture should be exposed, lest the active microscopic elements in it be set free and lodge in the wound. Diseased axillary glands should be taken away by

<sup>1</sup> *Postscript, September, 1867.*—The following story illustrates the difficulty of establishing the cancerous nature of the first disease. I have lately seen a lady whose breasts have both been amputated. The right mammary region is marked by two scars, and contains a soft fragment of the inner portion of the breast. At the axillary end of the scar which is in the place of the left breast, is recurrent Cancer. After the removal of the right mamma for reputed Cancer of a year's growth, four years elapsed, when there was recurrence below the middle of the scar, but not in the remnant of the breast. The new tumour was removed, and it proved to be a mere cyst. Thenceforth the right side remained healthy. Four years later, the left breast became cancerous, and within two more years that also was removed. The wound took fifteen months in healing, and was cancerous before it closed.

the same dissection as the breast itself, without dividing the intervening lymphatics; and the practice of first roughly excising the central mass of the breast, and afterwards removing successive portions which may be of doubtful soundness, should be abandoned. Only by deliberately reflecting the flaps from the whole mamma, and detaching it first at its edge, can the various undetected prolongations of the tumour and outlying nodules be included in the operation. To parts suspected of disease but not capable of removal it is desirable to apply the chloride of zinc. An edge of skin may be touched with the solid caustic; a paste of it may be laid on portions of the open wound; and, however healthy in appearance, the whole remaining surface may be washed with a solution of the chloride, of a strength proportionate to the delicacy or vitality of the textures and the thickness or thinness of the flaps, and varying from twenty to forty grains in the ounce of water. By these various applications the action of the zinc may be graduated to produce the strongest caustic effect or to merely whiten the superficial textures. Regard must be had to the depth of the subjacent structures in the use of the stronger preparations, especially on the wall of the chest; and in any strength the zinc should not be in contact for more than a moment with the large veins in the axilla, lest it should soak through their thin walls, and, producing phlebitis or a chemical action on the blood, should set up a traumatic pyæmia.

The conclusions, briefly stated, are partly theoretical, and partly practical. The former are—

That the recurrence of Cancer is due to local conditions:

That these conditions are not regional, so as to belong to structures out of continuity with the first tumour:

That neither are they organic, whether as indiscriminately involving the residue of a mamma operated upon, or so as to be transferable to the second breast in consequence of the removal of that first affected:

That, on the contrary, recurrent Cancer begins near the scar:

That, when free in both directions, it tends toward the axilla earlier than to the residue of the breast:

That, consequently, centrifugal dispersion, not organic origin, determines the recurrence of Cancer.

The practical conclusions are—

That Cancer of the breast requires the careful extirpation of the entire organ :

That the situation in which this operation is most likely to be incomplete is at the edge of the mamma next the sternum :

That, besides the breast, unsound adjoining textures, especially skin, should be removed in the same mass with the principal disease.

RECORD OF CASES  
TREATED IN THE  
LOCK HOSPITAL BY SYPHILISATION.

BY  
JAMES R. LANE, F.R.C.S.,  
AND  
GEORGE G. GASCOYEN, F.R.C.S.,  
SURGEONS TO THE LOCK HOSPITAL, ETC.

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THIS paper contains a brief report of the cases treated in the Lock Hospital by syphilisation, or the repeated inoculation of syphilitic matter in persons already the subjects of constitutional disease.

This peculiar method of treatment was originated by M. Auzias-Turenne about 1845, but met with so much ridicule and opposition from the French Academy of Medicine, that, except by the late M. Melchior Robert of Marseilles, it has scarcely again been tested in France. M. Sperino adopted it for a time in the venereal hospital of Turin, and he has published the result of a considerable number of cases submitted to this treatment, attended by varying success.

It remained, however, for Professor Böeck, of Christiania, to develop the system on a large scale, and a publication



by him in 1862, with another more recently by Dr. Bidentkap, giving the results of between 300 and 400 cases treated by syphilisation, has again directed attention to, and excited an interest in, this almost forgotten subject.

The great success claimed by these experimenters has popularised the treatment in their own country, and has created a desire in the profession generally to test the accuracy of their observations, and to watch the striking pathological phenomena which attend this novel method of treating constitutional syphilis.

The practice of syphilisation has evoked extreme hostility in England, and it had never been thoroughly carried out until the present series of cases was commenced under the superintendence of Dr. Böeck himself.

That gentleman, being on a professional visit to this country, most kindly acceded to the request of the Committee—then sitting to inquire into the treatment and prevention of venereal diseases in the Army and Navy—to state the result of his great experience; and he most generously, and at great personal sacrifice, offered to return to England for three months to initiate his system of treatment by syphilisation, provided he could obtain sufficient hospital accommodation for the purpose. At the request of the above Committee, and at the suggestion of the authors of this paper, the governors of the Lock Hospital very liberally placed some beds at his disposal, and the practice was commenced in September 1865, and continued under Dr. Böeck's supervision until the end of that year. After his departure from this country, the treatment of the cases begun by him was concluded by the authors, and some others have since been undertaken by them and carried through.

The opinion of Dr. Böeck with regard to the therapeutical action of syphilisation does not accord with that of MM. Auzias-Turenne and Sperino, who believe that the syphilitic poison is absorbed, and that the repeated inoculation of it produces a *saturation* of the system with syphilis, so that an immunity to the effects of the virus is at length acquired. Dr. Böeck considers that the introduction of the syphilitic

virus into the organism creates a *diathesis*, and is followed by a certain train of symptoms which succeed to each other with great regularity, but are usually prolonged over a considerable period of time. The syphilitic diathesis, when once established, cannot, he thinks, be increased or intensified by the further inoculation of virulent matter, but the continued insertion of this, by stimulating the existing disease, enables it to pass through its regular course, and therefore to complete the series of phenomena which follow the infection of the system by syphilis, in a far shorter time than if left to itself, or if subjected to any other method of treatment. Relapses and the occurrence of the so-called tertiary disease are therefore, he states, rarely found after this treatment; whilst the continued introduction of syphilitic matter into the system destroys, in a varying period of time, the receptivity of the patient, and ultimately produces a local as well as a general immunity to the disease.

This condition of immunity to the local action of the virus is gradually lost after the inoculations are discontinued; but the general immunity is very constant, and lasts as long as the duration of the diathesis—frequently throughout the life of the patient.

The method pursued by Dr. Böeck is as follows:—At the commencement three punctures are made on each side of the chest, and matter is inserted, derived either from a person who has a primary syphilitic ulcer, or from the artificial sores of a patient who is undergoing syphilisation.

After an interval of three days, if the punctures have developed pustules, three other inoculations are made from them in the same region of the body, and this process is repeated as long as pustules are produced; the inoculations being made at intervals of three days and the matter being always taken from the last formed pustules. When, at length, these are not inoculable fresh matter is employed, and the above process is repeated, until a positive result can no longer be obtained on the trunk. The same practice is then commenced on the arms, and continued there until the punctures fail, when a similar process is pursued on the thighs

until no more pustules result, and a condition of immunity more or less perfect is arrived at. In the ordinary run of cases this occurs in from three to four months.

Professor Böeck employs syphilisation for patients of all ages, even for very young infants who are the subjects of hereditary disease; in these latter, however, he has frequently found that the inoculations will not take: this often happens also in persons with constitutional syphilis who are suffering, at the same time, from some acute or debilitating disease. He has occasionally observed this temporary loss of inoculability when a syphilitic eruption has been ushered in with severe febrile disturbance, or has been preceded by a long-continued cachexia. After the disease has become more chronic, or the health has improved, the inoculations succeed and produce their curative effect, but these patients are more liable to relapses than those in whom the treatment has not been interrupted or delayed. If phagedæna occur, or should the health become impaired by excessive suppuration from the artificial ulcers, he does not discontinue the treatment, as he has found that the best remedy for such complications is to persevere with the inoculations. When the constitutional disease has existed for a length of time, the treatment by syphilisation is much prolonged, and relapses are more frequent: this is especially the case when the disease has reached its tertiary form, and Dr. Böeck recommends the use of iodide of potassium concurrently with inoculation.

A curious fact connected with syphilisation is, that not unfrequently a number of successive inoculations, which have remained without effect for many days and have been thought to be abortive, will suddenly and simultaneously develop pustules: this is supposed to depend upon some peculiar state of the system which has temporarily interfered with the receptivity of the patient.

Although Dr. Böeck considers that the two varieties of chancre are produced by the same virus, and that the result produced by their inoculation is essentially the same, he prefers the matter from an indurated sore for the purpose of

sypphilisation, as he has found that patients pass through the treatment and attain immunity more rapidly and satisfactorily when this is used, than when the secretion of a soft sore is inoculated.

According to the views of Dr. Böeck the administration of mercury interrupts and retards the natural course of syphilitic disease, and he believes that in like manner it interferes with its treatment by continued inoculation, diminishing therefore the efficacy of sypphilisation as a curative system. He was anxious in consequence, while in this country, to confine himself as much as possible to cases uncomplicated by a mercurial treatment, and this accounts in a great measure for the small number of patients treated by him at the Lock Hospital, for the great majority of those admitted with secondary disease are found to have been already subjected to a more or less complete mercurial course, either for the primary or secondary affection. Nearly all the cases admitted during the three months of Dr. Böeck's residence in England, which he considered suitable, were subjected to his treatment. Altogether, twenty-seven cases were treated in this manner; they were adults suffering from unmistakable constitutional syphilis, and in some the primary sore was still unhealed. In each instance, after full explanation, the sanction of the patient was obtained before commencing the inoculations, and they cheerfully and readily submitted to them.

In consequence of the differences observed in the progress of those patients who had previously been treated with mercury, and of the others who had not taken this drug, we have thought it better to divide the cases into two classes.

I. Those in which the disease was recent, and in which no mercury had been given.

II. Those which had been treated with mercury, and had suffered from one or more relapses after such treatment; amongst these was a very severe case of tertiary disease.

The first class comprises twenty-two, the second five cases.

We have endeavoured to record them as briefly as

possible, and have dwelt only upon such details as appeared to us necessary for the proper comprehension of the most important points involved.

We have stated in each the nature of the symptoms for which the treatment was undertaken; the progress of the inoculations; the effect of the treatment upon the symptoms; and the final result.

The inoculations practised on each patient were recorded by means of a diagram, three of which, illustrating cases 2, 8, and 9, are printed to show the method adopted; in addition to these a written diary of the patients was kept.

### CASES.

#### CLASS I.—*Cases which had not been treated with Mercury.*

CASE 1.—Female, C. J—, æt. 18, admitted August 17th, 1865, discharged February 20th, 1866.

*Symptoms.*—Incipient mucous tubercles on labia; squamous eruption on head, face, and trunk, with roseola on limbs, of five weeks' duration; multiple glandular enlargement in both groins. No previous treatment.

*Syphilisation.*—September 5th.—After two failures with matter from hard sores, the inoculations were commenced successfully with matter obtained from multiple soft sores in a male out-patient (same as in Cases 2 and 3). From these the inoculations were continued through seventeen successive generations on each side of the chest till October 23rd, when the pustules became too small and imperfect to reinoculate from.

October 26th.—Inoculations were made on the arms with matter originating in an indurated sore from a patient undergoing syphilisation, which was sent from Norway by Dr. Bidentkap, and it went through five generations. After this, matter was employed on the arms and thighs (chiefly from soft sores), and in one instance eleven generations were produced on the thighs, but for the most part they terminated at the second or third.

After the 27th December only very imperfect pustules were developed, and these never contained reinoculable matter. Immunity, therefore, may be said to have been attained in three months and twenty-two days.

*Progress.*—There was no improvement in the symptoms during the first two months of the treatment, but rather the contrary—fresh spots of eruption appeared, and the mucous tubercles on the labia increased in size. During the third month an amendment was apparent, and, by the time the inoculations would no longer take, the eruption had disappeared, as well as the mucous tubercles.

*Result.*—Left the hospital February 20th, 1866,—five months and fifteen days from the commencement of the treatment, and has not since been heard of. 281 inoculations were made; 157 of these were positive, 124 negative. The longest series of pustules obtained from the same matter was seventeen (soft).

CASE 2.—Female, M. A. B—, æt. 21, admitted August 17th, 1865, discharged February 13th, 1866.

*Symptoms.*—Indurated sore at inferior commissure, of six weeks' duration; eruption of roseola on trunk and upper extremities; multiple glandular enlargement in both groins and both posterior cervical regions. No previous treatment.

*Syphilisation.*—After two failures with matter from hard sores the inoculations were commenced successfully on September 5th, with matter from multiple soft sores in a male out-patient (the same as Cases 1 and 3), which went through fifteen generations on the chest and arms till October 20th. After this, matter from different sources, hard and soft, was inoculated through a gradually diminishing series on the trunk, arms, and thighs till December 13th, after which date no pustules were obtained which furnished reinoculable matter. Immunity therefore may be said to have been reached in three months and eight days.

*Progress.*—The local sore rapidly healed; the eruption was stationary for the first two months, but it had almost disappeared by the time the inoculations would no longer take.

*Result.*—She left the hospital for the Lock Asylum quite well, February 13th, 1866, five months and thirteen days from the commencement of the treatment. Her detention so long in the hospital was occasioned by the tediousness in healing of some of the inoculated sores on the lower extremities. She remained in the asylum in good health till March 1867, when she went to a situation. 276 inoculations were made; 155 of these were positive, 121 negative. The longest series of pustules obtained from the same matter was fifteen (soft).

This patient was inoculated by Mr. Lee on December 22nd to test her immunity. A small pustule was produced, but it was not reinoculable. (See Diagram I.)

**CASE 3.**—Female, R. B—, æt. 19, admitted August 17th, 1865, discharged January 30th, 1866.

*Symptoms.*—Mucous tubercles on labia; squamous eruption on trunk, neck, and arms, of recent date; glandular enlargement in both groins, and in both posterior cervical regions. No previous treatment.

*Syphilisation.*—September 5th.—The inoculations were commenced with matter from the same source as in Cases 1 and 2, and went through thirteen generations till October 11th. Matter from various sources, chiefly soft, was then inoculated on the arms and thighs till December 19th, but a series of five was the longest obtained.

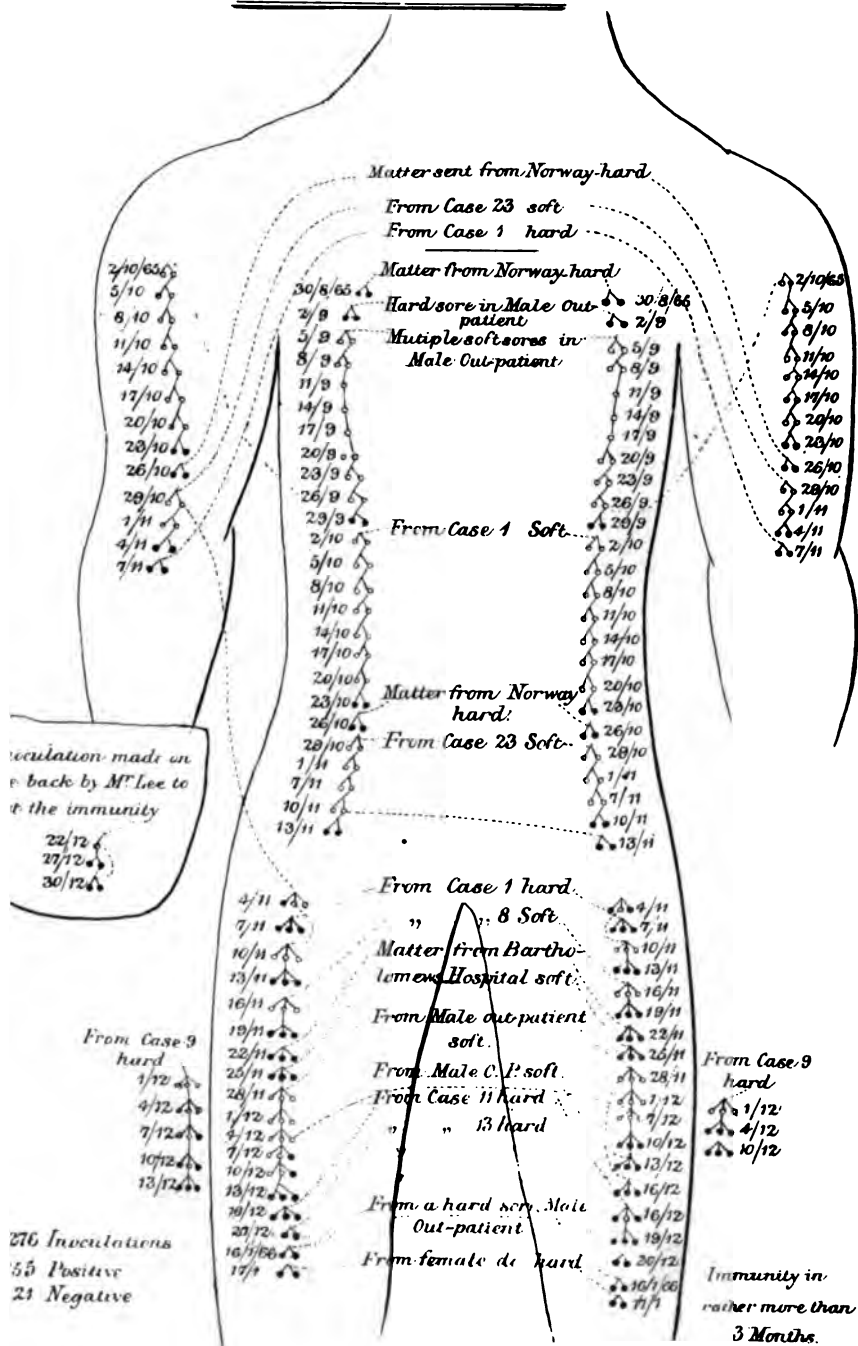
After this date no pustules were produced which secreted reinoculable matter. She was therefore considered, by Dr. Böeck, to have attained immunity.

This patient was also inoculated by Mr. Lee on December 22nd to test the immunity, and a small pustule was produced, which proved to be reinoculable once, but failed to reach a third series. She was inoculated four times subsequently with other matter, but no reinoculable pustules were obtained. An approximate immunity may therefore be said to have been established in about three and a half months.

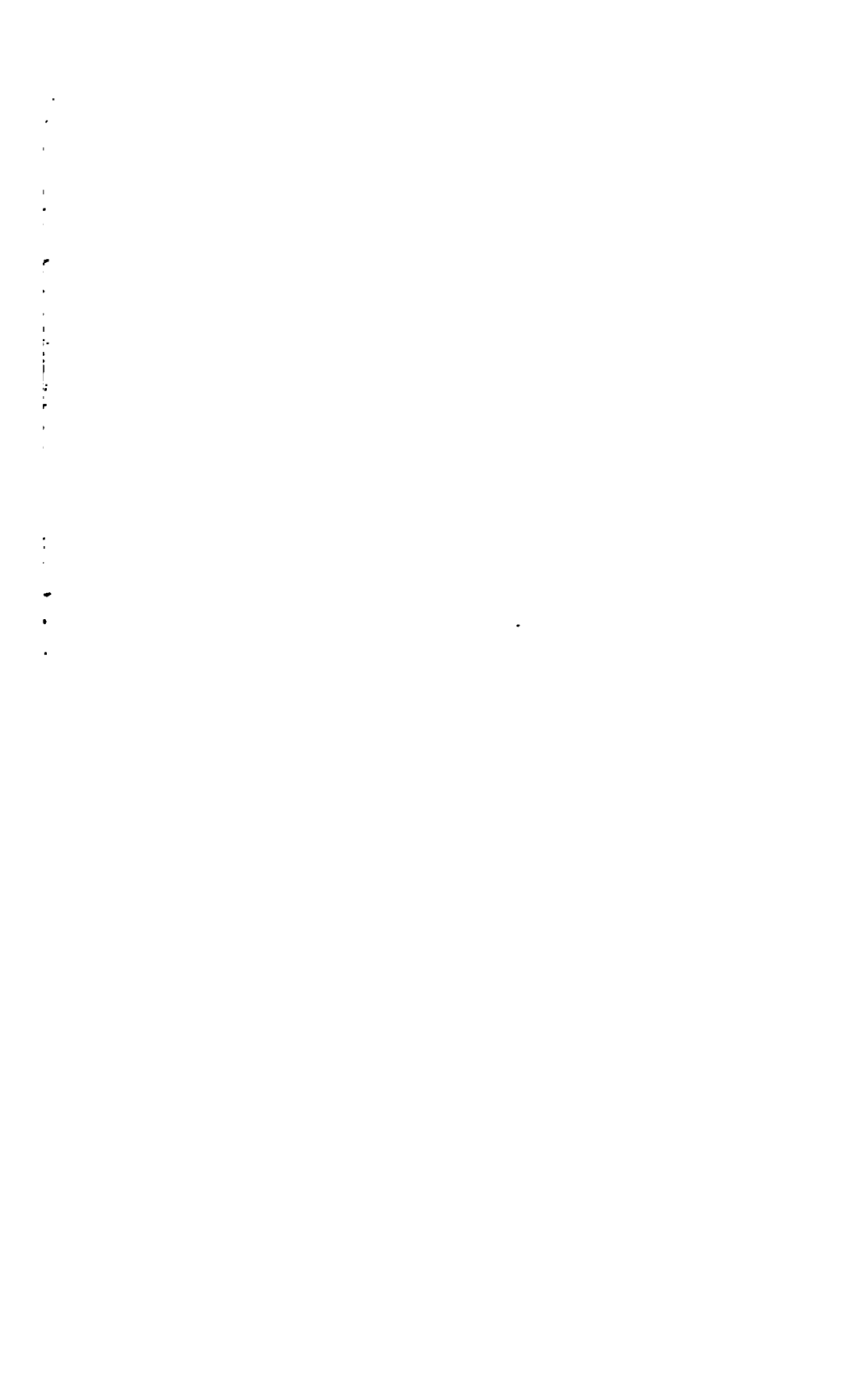
*Progress.*—By the time the inoculations were concluded all her venereal symptoms had disappeared.

*Result.*—She left the hospital quite well January 30th,

*Case 2.—Treatment Commenced 30<sup>th</sup> Sep<sup>r</sup> 1865.  
terminated 17<sup>th</sup> Jan<sup>y</sup> 1866.*







1866, five months from the commencement of the treatment, and has not since been heard of. 279 inoculations were made; 157 of these were positive, 122 negative. The longest series obtained from the same matter was thirteen (soft).

CASE 4.—Female, E. E—, æt. 21, admitted July 25th, 1865; discharged June 21st, 1866.

*History and Symptoms.*—This patient was admitted with a large sloughing sore on the left labium, which extended into the thigh and groin, and was of so severe a character that her life was for some time in danger.

When the sloughing had ceased and the ulcer had nearly healed, a well-marked secondary eruption appeared on her chest and back, accompanied with superficial ulceration of the tonsils; there was also enlargement of the inguinal and posterior cervical lymphatic glands.

*Syphilisation.*—September 21st.—The inoculations were commenced on the chest with matter from one of the inoculated sores in Case 2 (soft), which produced eight generations of good pustules, and was followed by matter from Case 23 (soft), which went through thirteen generations, terminating November 25th. After this date matter from various sources, hard and soft, was used on the arms and thighs till December 27th, when sloughing recommenced in the cicatrix of the sore for which she was originally admitted; it was therefore thought better to discontinue the treatment, especially as one of the neighbouring inoculations had taken on the same action.

The sloughing in the groin again spread to an alarming extent, and continued for some weeks, when at length it ceased and the wound slowly healed. The inoculated sore on the thigh, above referred to, sloughed till it attained a diameter of about an inch and a half, but none of the other inoculations were affected.

*Progress and Result.*—The patient recovered very slowly from the second attack of sloughing, and was unable to leave the hospital till June 26th, nine months and five days from the commencement of the treatment. By that time all her

**Result.**—Improvement in the case occurred July 21st, 1906. Two months and eight days from the commencement of the injections, and the treatment was in a measure successful. April 1907, the injections were made. One of these was positive, the rest negative. The uterus came from the same source as the first one, which yielded three more injections.

The fact of her having three months' amenorrhea was taken for liver disease, in which it differs in connection with the irregular progress and some duration of the injections.

**Case 6.**—Female, F. S.—admitted September 29th, 1905; discharged February 1st, 1906.

**Symptoms.**—Multiple tubercles in each ovary of size of small pieces of meat, and found with pieces of bone of one osseous in structure. Involvement of ligaments and posterior cervical lymphatic glands.

**Syphilis.**—January 1901.—Improvement in the case with matter from Case 1 originating from a soft spot, which went through nine generations failing in December 1901. The same matter was transferred to the arm in December 19th, and from thence to the thigh in December 1st, going through nine generations and failing in December 19th.

Inoculations were then performed with matter from various sources in January 1902, but a source of test was the same that could be obtained, and five consecutive attempts on the right thigh proved negative.

Finally, in January 1902, small pustules were produced with fresh matter from soft spots in a new inoculation. The result of these inoculations was not successful, as the patient left the hospital and did not again report. An opportunity for further treatment was obtained in this case in three months and twenty-three days.

**Progress.**—About a fortnight after the commencement of the treatment she had a sharp attack of pain in the right eye. This subsided under simple treatment by anodyne and sedatives, without any permanent damage to the eye.

The eruption and mucous tubercles continued, appearing

secondary symptoms had disappeared, the wound in the groin had healed, but the sore on the thigh, which had sloughed, was still covered with a scab about the size of a shilling.

She has been under observation to this date, April 1867, and has remained perfectly well. 259 inoculations were made; 181 of these were positive, 78 negative. The longest series from the same matter was twelve (soft).

The treatment by inoculation lasted three months and six days, but was interrupted before immunity was reached.

CASE 5.—Female, L. M—, æt. 25, admitted September 14th, 1865; discharged July 31st, 1866.

*Symptoms.*—Squamous eruption on face, with roseola on body and limbs, of recent date; tubercular patches on tonsils; indurated chancre at inferior commissure; indurated lymphatic glands in inguinal and posterior cervical regions. Has had no treatment for this disease, but says she took mercury for a liver complaint some years ago.

*Syphilisation.*—September 23rd.—The inoculations were commenced on the chest with matter from Case 2 (soft), which went through sixteen generations, and failed on November 10th; this was followed with matter from Case 8 (soft), which ceased to take at the fifth generation, November 25th. She was inoculated on the arms with matter from both hard and soft sores from October 23rd to December 31st. A first series of six generations was reached, but afterwards not more than two or three could be obtained, and the inoculations frequently failed.

She was inoculated on the thighs from November 19th to May 30th, the punctures taking freely, the final series, with matter from Case 4 (soft), extending through as many as thirty-eight generations. The treatment occupied a period of eight months seven days.

*Progress.*—The secondary eruption and other symptoms gradually disappeared, but during the treatment mucous tubercles appeared on the vulva on two occasions, which subsided under local applications.

*Result.*—Discharged to the Lock Asylum, July 31st, 1866, ten months and eight days from the commencement of the inoculations, and has remained well up to the present date, April 1867. 422 inoculations were made; 296 of these were positive, 126 negative. The longest series from the same matter was the final one, which reached thirty-eight generations (soft).

The fact of her having taken mercury some years before, for liver disease, is worthy of notice in connection with the irregular progress and long duration of the inoculations.

CASE 6.—Female, F. S—, æt. 18, admitted September 28th, 1865; discharged February 3rd, 1866.

*Symptoms.*—Mucous tubercles on labia; eruption of squamous patches on head, face, and trunk, with roseola on limbs, of one month's duration. Induration of inguinal and posterior cervical lymphatic glands.

*Syphilisation.*—October 11th.—Inoculated on the chest with matter from Case 5, originating from a soft sore, which went through sixteen generations, failing on November 28th. The same matter was transferred to the arms on November 19th, and from thence to the thighs on December 1st, going through nine generations and failing on December 19th.

Inoculations were then practised with matter from various sources till January 31st, but a series of two was the most that could be obtained, and five consecutive attempts on the right thigh proved negative.

Finally, on January 31st small pustules were produced with fresh matter from soft sores in a male out-patient. The result of these, however, was not watched, as she left the hospital and did not again attend. An approximate immunity was obtained in this case in three months and twenty-three days.

*Progress.*—About a fortnight after the commencement of the treatment she had a sharp attack of iritis in the right eye. This subsided under simple treatment by atropine and sedatives, without any permanent damage to the eye.

The eruption and mucous tubercles remained stationary

for more than two months, but they had completely disappeared by the end of January. She was much out of health when admitted, but improved greatly in appearance and gained flesh during her stay in the hospital.

*Result.*—Discharged February 3rd, 1866, three months and twenty-three days from the commencement of treatment, apparently quite well, and has not been heard of since. 260 inoculations were made; 154 of these were positive, 106 negative. The longest series obtained from the same matter was twenty-one (soft).

**CASE 7.**—Female, A. C—, æt. 18, admitted October 12th, 1865; discharged February 20th, 1866.

*Symptoms.*—Mucous tubercles on labia; eruption of well-marked roseola on body and limbs, which has existed four weeks; enlargement of inguinal and posterior cervical lymphatic glands. No previous treatment.

*Syphilisation.*—October 14th.—Inoculated on the chest with matter from Case 2 (soft), which went through eight generations. This was followed on November 7th with matter from Case 23 (soft), which also failed at the eighth generation; it was transferred on November 25th from the sides to the arms, but ceased to take at the third series.

On December 10th she was inoculated on the thigh with matter taken by Mr. Gascoven from a well-developed indurated sore in a female out-patient at St. Mary's Hospital, who afterwards had secondary symptoms. This took well, and produced eleven generations on the thighs, whence it was transferred to the arms, where it went through seven generations.

Between January 13th and February 7th she was inoculated four times—once with matter from a suppurating bubo, which failed at the second inoculation; and three times with matter originating in hard sores which had passed through patients undergoing syphilisation, but on each occasion with a negative result.

A practical immunity was attained in this case in three months and seventeen days.

*Progress.*—At the end of two months the eruption was

fading and the mucous tubercles were nearly well. By the time the inoculations were concluded there were no traces remaining of her venereal symptoms.

*Result.*—Discharged to the Lock Asylum February 20th, 1866, four months and six days from the commencement of treatment, and has remained there quite well up to the present date, April 1867. 265 inoculations were made; 160 of these were positive, 105 negative. The longest series obtained from the same matter was ten (hard).

CASE 8.—Female, H. W—, æt. 17, admitted October 12th, 1865; discharged January 28th, 1866.

*Symptoms.*—Copious and well-developed squamous eruption on trunk and extremities, of recent date; extensive and severe mucous tubercles on vulva, extending to the groin and inside of the thighs; multiple glandular enlargement in inguinal and posterior cervical regions. No previous treatment.

*Syphilisation.*—November 1st.—Inoculated with matter (hard) from Case 1, which failed. November 4th, inoculated on the chest with matter from Case 25 (soft), which produced eighteen generations of good pustules to December 25th. December 1st, inoculated on the arms from one of the above pustules, where twelve generations were obtained, to January 4th. On December 16th the matter was transferred from the arms to the thighs, where it went through seven generations to January 3rd.

Between January 6th and 22nd she was inoculated five times with matter from different sources, both hard and soft, but without success, the last time from a suppurating bubo, which took freely on other patients.

There was complete immunity, therefore, in this case in two months and three days.

*Progress.*—At the end of November the mucous tubercles were better, probably from the effect of local applications; the eruption was much the same. December 31st.—Eruption rapidly fading; local symptoms also much better.

*Result.*—January 28th.—Apparently quite well in every respect; discharged to the Lock Asylum two months and

twenty-eight days from the commencement of the treatment ; she remained there quite well till April 1867, when she left for a situation. 262 inoculations were made ; 168 of these were positive, 94 negative. Matter (soft) from one source only was employed ; it was never renewed, and went through twenty-one generations. (See Diagram II.)

CASE 9.—Female, M. A. S—, æt. 25, admitted September 25th, 1865 ; discharged April 10th, 1866.

*Symptoms.*—Mucous tubercles on labia, severe ; secondary eruption of roseola, mixed with squamous patches on chest and back, of a month's duration ; multiple glandular enlargement in both groins. No previous treatment.

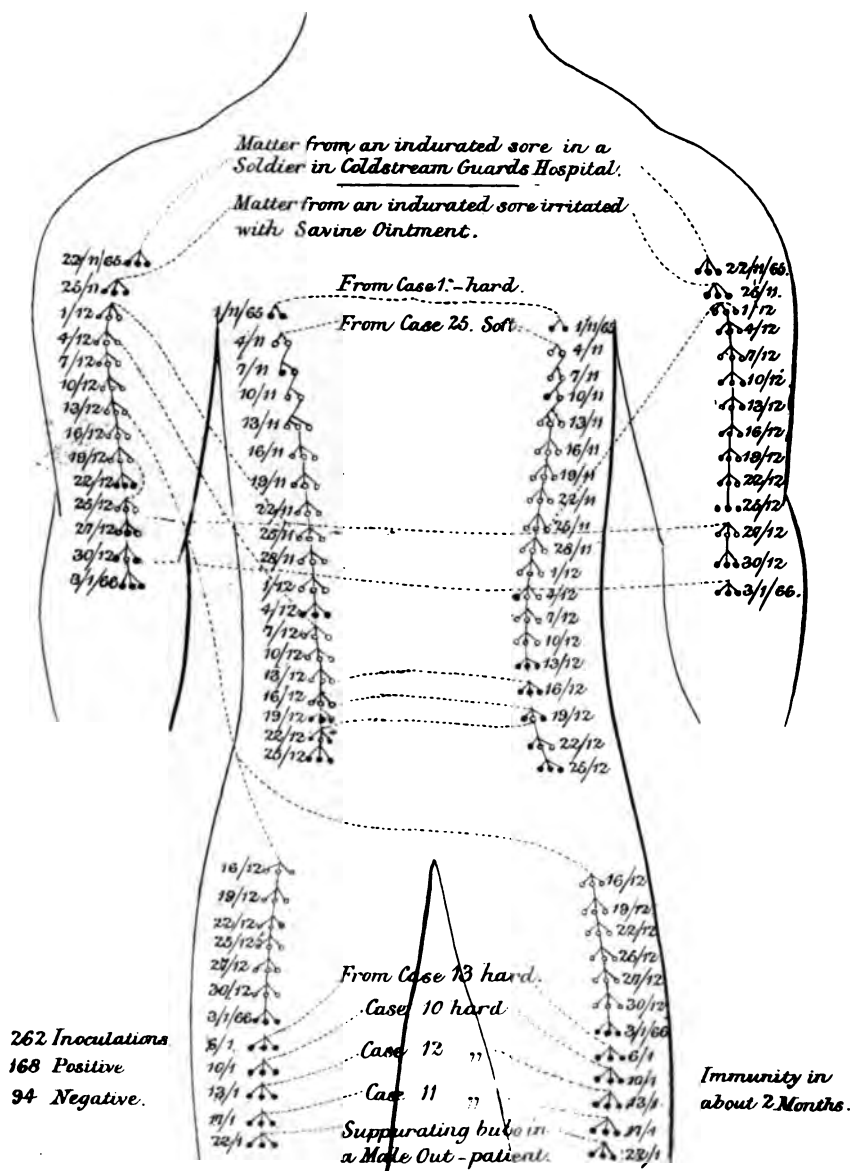
*Syphilisation.*—November 22nd.—No improvement having taken place in two months under simple and non-mercurial treatment, she was inoculated on the chest and also on the right arm with matter from Case 4, which had originated in a hard sore. The result was negative on the chest, but positive on the arm, where it went through nine generations.

November 25th.—Inoculated with negative result on left arm, with matter from an unirritated hard sore in a male out-patient ; also on the same day with matter from a characteristic hard sore in another male-patient (Case 22) which had been irritated for two days with savine ointment. Six punctures were made from this, three on each side of the chest. All six took freely, and produced well-developed pustules, which went through eleven generations to December 25th.

February 2nd.—After six consecutive failures on the thighs with matter from various sources, hard and soft, she was inoculated on the *right* thigh with matter from multiple soft sores in a male out-patient, and on the *left* thigh with matter from a well-marked indurated sore in another male out-patient, which had been irritated for two days with Unguentum Sabinæ. Large and well-marked pustules resulted on both thighs, and their appearance was in every respect identical. Both went through nine generations, and terminated on the same date March 3rd. After this no positive result could be obtained.



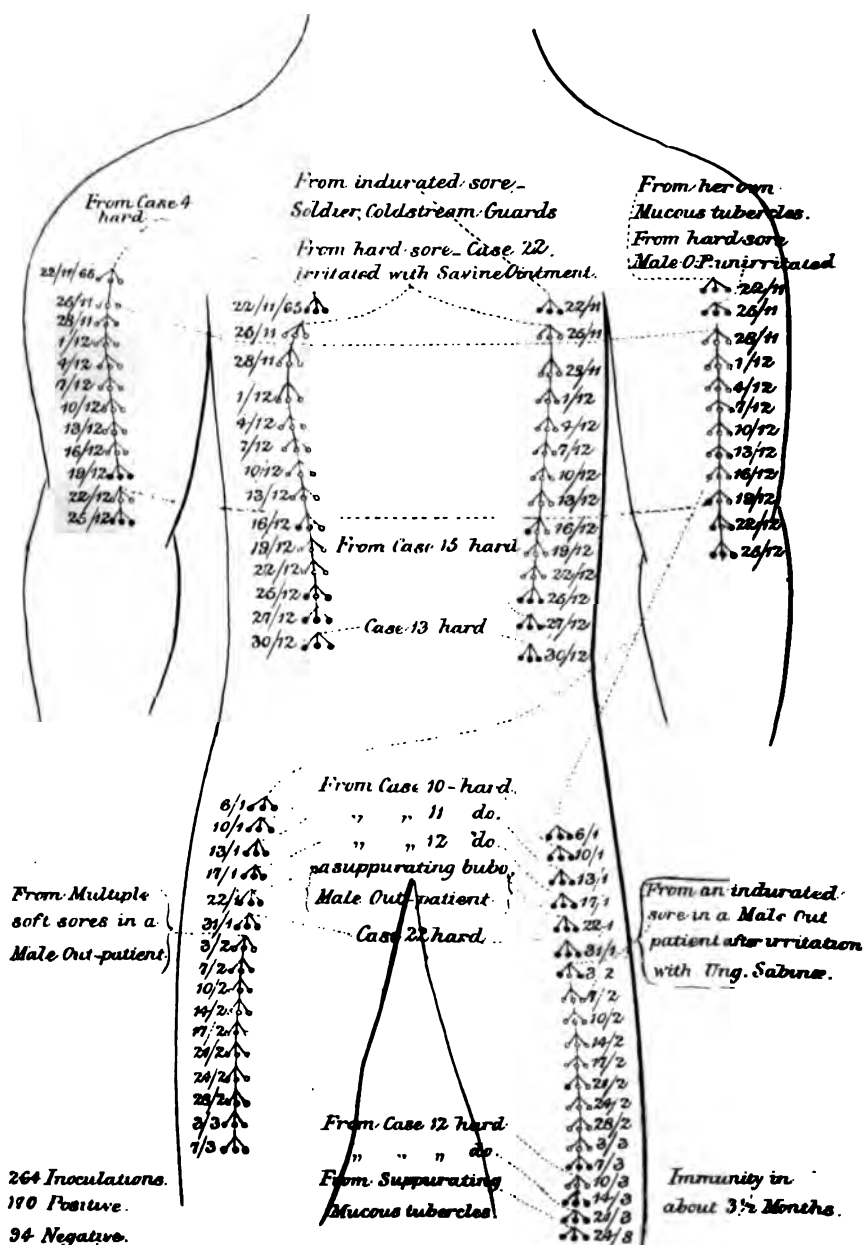
*Case 8.—Treatment Commenced Nov.<sup>r</sup> 1<sup>st</sup> 1865,  
terminated Jan<sup>y</sup> 22, 1866.*







*Case 9.—Treatment commenced Nov<sup>r</sup> 22<sup>nd</sup> 1865.  
terminated March 24<sup>th</sup> 1867.*



There was immunity in this case in three months and twenty days.

*Progress.*—There was a gradual subsidence in the syphilitic symptoms, which had entirely disappeared by the time the inoculations were concluded. Some of the later inoculations were very tedious in healing.

*Result.*—Discharged quite well, April 10th, 1866, four months and eighteen days from the commencement of treatment. 264 punctures were made; 170 of these were positive, 94 negative. The longest series from the same matter was eleven (hard).

She applied in March 1867, with swelling and superficial ulceration of left labium, which subsided in a fortnight under simple treatment. As she presented no other symptom, this was probably not connected with her former disease. (See Diagram III.)

CASE 10.—Female, E. F—, æt. 19, admitted November 11th, 1865; discharged April 3rd, 1866.

*Symptoms.*—Large raised patches of mucous tubercles on labia, extending to the right groin; well-marked eruption of roseola on chest and back, of recent date; induration of inguinal and posterior cervical lymphatic glands. No previous treatment.

*Syphilisation.*—November 28th.—Inoculated with matter from soft sores in a male out-patient, which went through seven generations; this was followed by matter (hard) from Case 7, which failed at the second generation; then by matter (also hard) from Case 12, which failed at fourth generation.

December 22nd to February 3rd.—Inoculated on arms with matter from various sources, principally hard, but none went through more than four generations.

February 10th.—Inoculated on both thighs from Case 9, on the left side with matter originating in a hard sore, on the right side in a soft sore. As in Case 9, there was no difference in the appearance of the pustules, which were all large and well developed; the “soft” matter failed at the seventh generation, but the “hard” reached the fourteenth.

The inoculations occupied altogether more than four months, failing on March 31st.

*Progress.*—By the end of December, one month from the commencement of the inoculations, the mucous tubercles were improving. At the end of January they were nearly well, and the eruption also was fading. At the end of March there was no trace of either.

*Result.*—Discharged, apparently quite well, April 2nd, 1866, four months and four days from the commencement of treatment; she has not since been heard of. 244 inoculations were made; 127 of these were positive, 117 negative. The longest series from the same matter was thirteen; this was the final series, and was from "hard" matter.

CASE 11.—Female, E. N—, æt. 20, admitted October 6th, 1865; discharged April 3rd, 1866.

*Symptoms.*—November 28th.—Mucous tubercles on labia; copious eruption of roscola, mixed with patches of psoriasis on trunk and limbs; induration of inguinal and posterior cervical lymphatic glands.

Admitted in October with sores on labia, which soon healed. Three weeks afterwards the eruption appeared, and later the mucous tubercles on the vulva. No previous mercurial treatment.

*Syphilisation.*—November 28th.—Inoculated on the chest with matter from Case 4 (hard), which went through five generations to December 10th; and was followed by matter of the same character from the chest of Case 9, which went through nine generations to January 13th.

November 28th to March 3rd.—Inoculated on the arms twenty-one times with matter from various sources, hard and soft, but no pustules were obtained which could be reinoculated. She was inoculated on the thighs six different times with matter originating in indurated chancres, but with a negative result. This patient was successfully inoculated with matter which had origin in hard sores only. Immunity was obtained in six weeks, after fourteen consecutive inoculations—in one series of five and in another of nine generations.

*Progress.*—The venereal symptoms gradually disappeared, and she was entirely free from them by the end of March.

*Result.*—Discharged, apparently quite well, April 3rd, 1866, four months five days from the commencement of the inoculations. Attended as an out-patient on May 5th, with a small patch of mucous tubercles near the anus; this shortly got well under the application of nitrate of silver and astringent lotions; she has not been heard of since. 266 inoculations were made; 82 of these were positive, 184 negative. The longest series obtained from the same matter was nine (hard).

CASE 12.—Female, H. C—, æt. 18, admitted November 2nd, 1865; died of sloughing phagedæna May 24th, 1866.

*Symptoms and History.*—Was admitted with a large sloughing sore on the right labium, extending to the thigh; both labia were covered with patches of mucous tubercles in a state of ulceration. Copious eruption of roseola mixed with squamous patches; induration of inguinal and posterior cervical lymphatic glands. First noticed disease six weeks before admission; after three weeks went to the workhouse, and whilst there the sloughing commenced. After a month's treatment in the Lock Hospital the sloughing sore was nearly well, but the secondary affections remained in the same condition, though her health had very much improved. No previous mercurial treatment.

*Syphilisation.*—Commenced December 1st. Was inoculated six times to December 10th, negatively, with matter from Case 4 (hard).

December 11th.—A positive result was obtained with matter from the arm of Case 9 (hard), which went through eighteen generations of well-developed pustules on the sides and arms to February 10th.

February 14th.—Inoculated on the arms with matter from the left thigh of Case 9 (hard), which went through twenty-one generations on the arms and thighs to May 6th, when it was still taking freely, but was discontinued, in consequence of the reappearance of sloughing in the cicatrix on the labium,

five months and five days after the commencement of the inoculations.

*Progress and Result.*—Her general health was at first very indifferent, but improved greatly, and during the first four months she gained strength and flesh ; towards the end of that time the mucous tubercles were well and the eruption was nearly gone.

On April 11th the sloughing of the labium recommenced, and in spite of all treatment spread to the thighs and lower part of the abdomen ; she gradually sank, and died May 24th, 1866, five months and twenty-four days from the commencement of the inoculations. No sloughing took place at any of the inoculated spots. 285 inoculations were made ; 196 of these were positive, 89 negative. The longest series obtained from the same matter was twenty-one (hard) ; no matter from soft sores was used in this case.

CASE 13.—Female, A. II—, æt. 21, admitted December 7th, 1865 ; discharged June 26th, 1866.

*Symptoms.*—Mucous tubercles on labia of three weeks' duration, and similar patches on tonsils ; eruption of squamous patches mixed with roseola on chest and back ; induration of inguinal and posterior cervical lymphatic glands. No previous mercurial treatment.

*Syphilisation.*—December 13th.—Inoculated with matter from the arm of Case 9 (hard), which went through nine generations to January 6th.

About this date she was attacked with jaundice, which lasted for a month (till February 7th). During this time several inoculations were made with matter from different sources, hard and soft, but the result was uniformly negative.

February 10th.—Having recovered from the jaundice, she was inoculated on the right arm with matter from the left thigh of Case 9 (hard), which took freely and went through thirteen generations on the arms and thighs till March 28th. At the same time the left arm was inoculated from soft sores in a male out-patient, and seven generations resulted, the pustules on the two arms being identical in appearance. After these had failed, matter from Case 22



(hard) went through six generations on the thighs from April 21st to May 12th.

From this date, the pustules obtained were small and never passed beyond the third generation. From May 26th to June 9th five inoculations failed altogether; immunity was obtained in five months and ten days.

*Progress.*—The symptoms gradually disappeared; her health was good throughout, except while she was suffering from the jaundice above mentioned.

*Result.*—Left the hospital for the Lock Asylum June 26th, 1866, six months and thirteen days from the commencement of treatment, and she remained there quite well for six months. 287 inoculations were made; 164 of these were positive, 123 negative. The longest series obtained from the same matter was thirteen (hard).

CASE 14.—Female, M. L—, æt. 18. admitted December 7th, 1865; discharged April 3rd, 1866.

*Symptoms.*—Mucous tubercles on labia; diffused squamous eruption on trunk and limbs, of recent date. Induration of inguinal and posterior cervical lymphatic glands. No previous mercurial treatment.

*Syphilisation.*—December 13th.—Inoculated with matter from Case 9 (hard), which went through five generations; and was followed by matter from Case 5 (hard), which failed at the third generation.

January 6th to February 7th.—Some imperfect pustules resulted, but more frequently failures, with matter from various sources, hard and soft.

February 10th to March the 3rd.—Inoculated seven different times on the arms, with four partially successful results and three failures.

March 3rd to March 28th.—Seven inoculations were made on the thighs with matter from various sources, but two only produced pustules; these, however, could not be reinoculated. Immunity appeared to be complete in two months and twenty-seven days.

*Progress.*—Uniformly good; the syphilitic symptoms

gradually disappeared, and were quite well when the inoculations were discontinued.

*Result.*—Discharged to the Lock Asylum April 3rd, 1866, three months and twenty-one days from the commencement of treatment; she has remained well to the present date, April 1867. 173 inoculations were made; 66 of these were positive, 107 negative. The longest series from the same matter was five (hard).

CASE 15.—Female, E. D—, æt. 16, admitted November 9th, 1865; discharged June 27th, 1866.

*Symptoms.*—Mucous tubercles in a state of ulceration on labia and around anus; severe squamous eruption on trunk; scattered patches of the same on limbs; face covered with thick scaly patches, of about six weeks' duration. No previous mercurial treatment.

*Syphilisation.*—December 16th to January 31st.—Inoculated on the trunk with matter originating in hard sores; the pustules were small and often failed altogether; they only once reached five generations.

February 7th.—Good pustules obtained on the arms with matter from a soft sore; this went through nine generations to March 7th, and was followed by matter from Case 12 (hard), which went through six generations to March 28th.

March 31st.—Inoculations commenced on the thighs; after several failures, a series of six generations was obtained with matter from an out-patient (soft), terminating May 9th; no good pustules were again produced, and from May 23rd to June 19th the result of five inoculations with matter from different sources was altogether negative; the last of these was made with matter from soft sores, which took freely on the bearer. Immunity complete in four months and twenty-seven days.

*Progress.*—For three months there was no improvement in the eruption on the body, whilst that on the face became much worse. In April she was attacked with iritis in both eyes; this, however, subsided in about three weeks under atropine drops and mild astringent lotions. At the end of

April the eruption began to fade, and by the middle of June, with the exception of some trifling stains, it was altogether gone.

*Result.*—Discharged to the Lock Asylum June 27th, 1866, six months and eleven days from the commencement of the inoculations, and has remained there well to the present date, April 1867. 262 inoculations made; 131 of these were positive, 131 negative. The longest series from the same matter was nine (soft).

CASE 16.—Female, E. T—, æt. 18, admitted June 21st, 1866; discharged December 17th, 1866.

*Symptoms.*—Large crop of mucous tubercles on labia; well-developed squamous eruption on arms and neck, of recent date; slight enlargement of posterior cervical glands, none of those in the groin. Previous treatment local only.

*Syphilisation.*—June 30th.—Inoculated with matter taken direct from soft sores in another female patient, which went through eleven generations to August 4th.

August 11th.—Inoculated with matter from a very well-marked typical hard sore on the labium of a female in-patient. Six punctures were made, five of which failed, but one produced a pustule, from which a series of thirteen generations was obtained, to September 26th.

September 29th.—Inoculated on the arms with matter from Case 17 (soft); this went through ten generations to November 1st.

November 4th to December 3rd.—Inoculated on the thighs with the same matter, and also from Case 18; the pustules were either not reinoculable or the punctures failed altogether. Immunity in about five months.

*Progress.*—Symptoms remained stationary for the first two months, after which they gradually disappeared, and were entirely gone some time before the inoculations were concluded.

*Result.*—Discharged to the Lock Asylum December 17th, 1866, five months and seventeen days from the commencement of treatment; has remained there quite well to the

present date, April 1867. 156 punctures were made; 116 of these were positive, 40 negative. The longest series from the same matter was thirteen (hard).

CASE 17.—Female, E. H—, æt. 19, admitted June 21st, 1866; discharged April 29th, 1867.

*Symptoms.*—Mucous tubercles on labia; squamous eruption on trunk and lower extremities of about two months' duration; indurated inguinal glands. No previous treatment.

*Syphilisation.*—June 30th.—Inoculated on the chest with matter taken direct from soft sores in a female in-patient. This went through twenty-seven generations of good pustules till October 2nd.

September 29th.—Inoculated on the arms with matter from Case 16 (hard), which went through eleven generations to November 4th, and was then transferred to the thighs, where it went through six generations to November 23rd, at which date it failed altogether, the pustules having been for some time very imperfect.

November 30th.—She was inoculated by the house-surgeon, but not by our direction, from a patient with a spreading phagedænic sore on the labium and buttock; two punctures were made on each thigh, and large pustules having been developed, two more inoculations were made from them three days later. Almost immediately all these punctures took on a phagedænic action, and spread to a very serious extent. Those on the left thigh coalesced and produced a sloughing sore of considerable size. The sloughing action was not arrested for some weeks; she then recovered slowly, but was not in a condition to leave the hospital till April 29th, 1867.

*Progress.*—Her syphilitic symptoms showed no improvement for the first two months, after which they gradually subsided, and were scarcely perceptible at the end of four and a half months, when the inoculations were discontinued. At that time she was approaching a condition of immunity to ordinary matter, the pustules had been for some time very imperfect, and twice had failed altogether,

when the unfortunate application of matter from a phagedænic sore was made.

*Result.*—Discharged April 29th, 1867, ten months from the commencement of the inoculations. She was quite well of her syphilitic symptoms, but had suffered greatly in health from the effect of the sloughing sores. 137 inoculations were made; 122 of these were positive, 15 negative. The longest series from the same matter was twenty-seven (soft).

CASE 18.—Female, M. R—, æt. 38, admitted July 12th, 1866; discharged February 5th, 1867.

*Symptoms.*—Mucous tubercles on vulva, ulcerated; mucous patches on tonsils; squamous eruption on trunk and limbs, of about a month's duration; enlargement of inguinal lymphatic glands. No previous mercurial treatment.

*Syphilisation.*—July 21st.—Inoculated with matter from Case 17 (soft), which failed at the third generation, but being repeated from this same source went through twenty-six generations, viz. sixteen on the trunk and ten on the arms.

November 1st.—Inoculated on the thighs with matter from Case 16 (hard), which went through eight generations to November 23rd; also with matter from Case 17 (hard), which went through four generations, but these pustules were very small and imperfect.

November 30th.—This patient was also inoculated by the house-surgeon with matter from the same phagedænic sore as that used in Case 17. The result was similar; the pustules rapidly took on a sloughing action, and spread to about the size of a penny-pie. After the slough separated the sores were a long time healing. The sloughing, however, was much less extensive in this than in the previous case. These sores caused her detention in hospital till February 5th, 1867. There was approximate immunity to the action of ordinary matter in about four months.

*Progress.*—No improvement in the eruption for more than two months. On September 25th she was attacked with iritis. For this she was treated at first, like the other two cases, with atropine drops and sedative astringent lotions,

but in a few days, the inflammation having increased, and the loss of the eye being threatened, mercury was given until her gums were slightly touched. Under this treatment the iritis rapidly subsided and her eye suffered no permanent damage. Her other syphilitic symptoms now gradually disappeared, and she was apparently quite well, so far as they were concerned, some time before she left the hospital, where she was detained longer than would otherwise have been the case in consequence of the sloughing ulcers.

*Result.*—Left the hospital well February 5th, 1867, six months and fifteen days from the commencement of treatment. 130 inoculations were made; 103 of these were positive, 27 negative. The longest series obtained from the same matter was twenty-six (soft).

CASE 19.—Female, H. F—, æt. 18, admitted February 8th, 1866; left by her own wish May 18th, 1866.

*Symptoms.*—Swelling and excoriation of external genitals, with vaginal discharge; squamous eruption on face, trunk, and limbs, of five weeks' duration. No previous treatment.

*Syphilisation.*—February 14th, 1866.—Inoculated with matter from Case 9 (hard), which failed at the fourth generation. She was then inoculated on the trunk, till March 28th, with matter from Case 12 (hard), but a series of three was the longest that could be obtained.

March 31st to April 18th.—Inoculated on the arms with matter from different sources; result always negative.

April 21st.—Inoculated with matter originating in a hard sore; this had reached eight generations on May 17th and was still taking freely, when she left the hospital on leave for the day, and did not return. The inoculations were continued for three months and three days; there was as yet no evidence of approaching immunity.

*Progress.*—During the three months she was under treatment, there was no improvement in the eruption for the first six or seven weeks, after which time it appeared to be slowly fading away. The excoriations on the labia soon got well,

but were followed by a crop of mucous tubercles shortly afterwards.

*Result.*—Left the hospital May 17th, 1866, before the inoculations were concluded, three months and three days from their commencement. 102 inoculations were made, 74 of these were positive, 28 negative. The longest series obtained from the same matter was eight (hard).

**CASE 20.**—Male, D. B—, æt. 22, a Swedish sailor, admitted December 7th, 1865; discharged by his own wish, June 19th, 1866.

*Symptoms.*—Large and well-marked indurated sore on prepuce; eruption of roscola on trunk; mucous tubercles at umbilicus and anus; multiple glandular enlargement in groins and posterior cervical regions.

The sore was contracted four months ago; it appeared eight days after connexion and has never healed. No previous treatment.

*Syphilisation.*—December 6th.—Inoculated by Dr. Böeck at Guy's Hospital, the day before his admission, with matter from a soft sore. This went through fourteen generations of well-developed pustules on the chest, terminating January 20th.

January 6th.—Inoculated on the chest with matter from the right arm of Case 22 (the sore from which this matter was taken was the product of auto-inoculation from a typical indurated chancre on the prepuce); it went through thirty generations, terminating on May 15th.

April 21st.—Inoculated unsuccessfully with matter (hard) from Case 21.

May 19th to June 14th.—Inoculated repeatedly on both arms with matter from an in-patient with a well-marked indurated chancre, but without result.

June 9th.—Inoculated successfully on the chest from a suppurating bubo, but it ceased to take at the third generation.

The inoculations occupied a period of more than six months, but although the pustules were large and well developed for four or five months, immunity was not established when he left the hospital.

*Progress.*—The eruption had disappeared in about two months; the mucous tubercles at the umbilicus were very tedious in healing, though frequently touched with nitrate of silver; others broke out at the anus at different periods during the treatment.

*Result.* Discharged at his own request, June 19th, 1866, six months and thirteen days from the commencement of treatment. 352 inoculations were made; 178 of these were positive, 174 negative. The longest series obtained from the same matter was thirty (hard).

CASE 21. Male, R. M—, æt. 26, admitted January 4th; discharged May 8th, 1866.

*History and Symptoms.*—Contracted a sore on the prepuce three months ago, for which he has been attending as an out-patient for two months, but no mercury has been given. On admission there was induration of the prepuce, causing phimosis; well-marked eruption of roseola; enlargement of inguinal and posterior cervical glands.

*Syphilisation.*—January 13th.—Inoculated from the left arm of Case 22; the sore from which this matter was taken was the product of inoculation from a female patient (Case 9), in whom direct inoculation from the indurated chancre of the same Case (22) had been successful. This produced six generations. After some failures he was successfully inoculated on March 17th with matter from Case 20 (this also had its origin in the indurated chancre of Case 22); it went through thirteen generations and failed on May 1st.

*Progress and Result.*—The eruption was gradually fading, but had not entirely disappeared, nor was immunity obtained on May 8th, when he left the hospital by his own wish, after three months and twenty-five days' treatment. He has not since been seen. 133 inoculations were made; 93 of these were positive, 40 negative. The longest series obtained from the same matter was thirteen (hard).

CASE 22, under Mr. W. Coulson.—Male, C. D—, æt. 20, admitted as out-patient September 23rd, 1865, as in-patient



January 4th, 1866; discharged at his own request, April 6th, 1866. The treatment throughout was non-mercurial.

*History and Symptoms.*—The primary chancre appeared early in September, about a fortnight after connexion; it commenced as a flat dry sore behind the corona, and continued to furnish a scanty thin serous secretion until irritated with savine ointment.

November 23rd, 1865.—Large indurated sore, the size of half a crown, involving corona glandis, and adjacent part of prepuce on right side; well-marked eruption of roscola on head, face, arms, and trunk; multiple glandular enlargement in inguinal and posterior cervical regions.<sup>1</sup>

*Syphilisation.*—November 27th.—Was inoculated on the right arm from his own chancre, which had been irritated for several days with savine ointment. The result being negative, the inoculations were continued daily till December 15th, when one pustule was produced, from which a series of well-developed pustules was obtained, extending through ten generations to January 18th.

On the same day, viz. November 27th, he was inoculated on the right side of the chest from a soft sore, which went through twelve generations, and failed on January 18th.

January 21st.—Inoculated successfully with matter from Case 21 (hard); the sore from which this was taken had its origin in one resulting from direct inoculation of his own indurated chancre upon a female patient (Case 9). This failed at the third generation.

January 29th.—Inoculated from an indurated sore in a male out-patient, with positive result, but the pustules were only imperfectly reinoculable.

December 15th.—Was inoculated on the left arm with matter from Case 9, which had been originally derived from his own indurated chancre; this went through seventeen generations, terminating February 18th.

<sup>1</sup> On November 25th a female patient (Case 9) was inoculated from this man's chancre after it had been made to suppurate with savine ointment; six punctures were made, and all proved successful. This was after the sore had been irritated for two days with the ointment, and before he had been inoculated with matter from any other source.

March.—Was inoculated successfully from a hard sore in a male out-patient; this went through ten generations to April 4th.

On April 6th the inoculations were discontinued by his own wish, immunity not having been yet obtained after four months and ten days' treatment.

*Progress.*—His chancre healed very slowly, and the eruption remained for a considerable time. There was still a distinct mottling of the skin when he left the hospital, April 6th, 1866, four months and eighteen days from the commencement of the inoculations.

*Result.*—This patient applied at the hospital to Mr. Coulson a few months afterwards, with a return of his eruption. 279 inoculations were made; 173 positive, 106 negative. The longest series obtained was seventeen (hard).

*CLASS II.—Cases which had undergone a previous mercurial treatment.*

CASE 23.—Female, A. W—, æt. 23, admitted August 24th, 1865, discharged February 5th, 1866.

*Symptoms and History.*—Patches of squamous eruption on trunk, face, and head. Disease of six months' duration; was treated in the first instance with mercury to slight salivation, under which the symptoms disappeared; has had two relapses, and has each time taken mercury. The present is her third relapse.

*Syphilisation.*—October 5th, 1865.—Inoculations commenced with matter from Case 4 (soft), which went through eight generations of good pustules to October 29th.

October 29th to November 19th.—The pustules produced on the sides from different kinds of matter were very imperfect, and could not be made to reach a third generation.

November 11th to December 25th.—The same was the case on the arms, though matter from eight different sources was employed.

November 22nd to February 12th.—On the thighs also the inoculations were not followed by the usual development

of pustules. Once only a series of five generations was reached with matter from Case 10 (soft). With this exception a third series was never attained, though matter from sixteen different sources was used. There was an immunity nearly, if not quite, complete in about four months.

*Progress and Result.*—The secondary manifestations gradually disappeared, and she was apparently quite well when she left the hospital, on February 5th, 1866, four months from the commencement of treatment. She returned in a short time with a recurrence of the eruption; for this the bichloride of mercury was prescribed, and it speedily disappeared; she has not since been heard of. 273 inoculations were made; 92 of these were positive, 181 negative. The longest series obtained from the same matter was eight (soft).

CASE 24.—Female, M. G—, æt. 26, admitted September 19th, 1865, discharged April 10th, 1866.

*Symptoms.*—Mucous tubercles on labia and around anus; copious eruption of squamous patches on body and limbs; multiple glandular enlargement in inguinal and posterior cervical regions. Has had disease for three months and has been treated at the Middlesex Hospital by mercurial fumigations.

*Syphilisation.*—October 11th.—Inoculated with matter from Case 27 (soft), which went through ten generations on the chest to November 10th. After this no good pustules resulted from inoculation on this region of the body, though matter from several sources was employed.

November 13th to December 27th.—Inoculated on the arms with eight different kinds of matter, but a series of four was the longest obtained.

November 28th to March 4th.—Inoculated on the thighs with matter from seventeen different sources, six times with a positive result, but on one occasion only was a series of six generations reached. Eleven times the result was entirely negative. Immunity was obtained in five months.

*Progress.*—The eruption gradually disappeared and the mucous tubercles on the labia got well, but broke out again once or twice during the treatment; similar spots also made

their appearance in the mouth; in both situations they were treated by the occasional application of nitrate of silver.

*Result.*—She was discharged to the Lock Asylum quite well, April 10th, 1866, six months from the commencement of the treatment. She shortly afterwards left the asylum and has not since been heard of. 291 inoculations were made; 127 of these were positive, 164 negative. The longest series obtained from the same matter was ten (soft).

**CASE 25.**—Female, M. B—, æt. 25, admitted September 28th, 1865; discharged April 10th, 1866.

*Symptoms and History.*—Well-developed squamous eruption on face, trunk, and limbs; remains of mucous tubercles on labia. She noticed sores on the vulva five months before her admission; two months later the eruption appeared; has been treated with mercurial pills to salivation.

*Syphilisation.*—October 23rd, 1865.—Inoculated on the chest with matter from Case 2 (soft), which went through nine generations of pustules to November 19th, but no good pustules were afterwards obtained in this region.

November 4th to December 27th.—Inoculated on the arms with matter from Case 4 (hard); this went through twelve generations.

December 1st to April 7th.—Inoculated on the thighs with matter from a variety of sources, but a series of eight was the longest attained, and the failures were frequent. When the treatment was discontinued, it was very difficult to obtain pustules, and they were small and abortive, but there was not an absolute immunity.

*Progress.*—The eruption gradually subsided, but there were still some faint traces of it when she left. Some of the inoculated spots on the thighs had been very slow in healing, and were still covered with thick scabs.

*Result.*—Left the hospital April 10th, 1866, five months and eighteen days from the commencement of treatment. 339 punctures were made; 195 of these were positive, 144 negative. The longest series from the same matter was twelve (hard).

May 29th.—Was re-admitted with mucous patches at umbilicus and ulcers on the inside of the cheek, which soon disappeared under tonics and simple local treatment. The inoculated spots on the thighs were still covered with scabs.

She left the hospital quite well, July 10th, 1866, and has not attended since.

CASE 26.—Female, A. S—, æt. 23, admitted November 18th, 1865; died April 5th, 1866.

*Symptoms and History.*—Copious and well-developed squamous eruption on face and limbs; multiple enlargement of inguinal glands; severe nocturnal pains in head and limbs. Was in the hospital five months previously with the primary sore and a secondary eruption, for which she was treated with mercury. The eruption disappeared, but has recently returned in a more severe form. She is in failing health and much emaciated.

*Syphilisation.*—November 28th.—Inoculated with matter from Case 4 (hard), which failed. She was then inoculated on forty-one occasions till March 17th, a period of three months and nineteen days, eighteen times on the trunk, nineteen on the arms, and four times on the thighs. Every variety of matter at our disposal was used, but the result was almost invariably negative; once on the chest, and once on the arms, small pustules were obtained, which were reinoculable once, but not a second time.

*Progress and Result.*—Under iodide of potassium, wine, and good diet, she appeared to improve in health for a few weeks after the inoculations were first attempted, but at the end of that time an abdominal enlargement, occupying the right hypochondriac and epigastric regions, became evident. This tumour increased rapidly in size; it presented a smooth uniform surface, and was somewhat soft and elastic to the touch. Our impression was that she was suffering from malignant disease of the liver, and this opinion was strengthened by the rapid emaciation and general cachexia which accompanied the growth of the tumour.

She gradually sank and died April 6th, 1866, four months after the first appearance of the swelling. The post-mortem examination showed an enormous enlargement of the liver, which was affected uniformly throughout with amyloid degeneration; the other organs were healthy; the secondary eruption had not disappeared at the time of her death.

This case is of peculiar interest, in consequence of the almost uniform failure of the inoculations. They were continued for the sake of experiment, but it seems clear that they had no influence over the progress of her case one way or the other. 248 inoculations were made; 226 of these were negative, 22 are marked as positive, but they were so small and imperfect that they scarcely deserve to be so called.

CASE 27.—Female, J. S—, æt. 33, admitted September 8th, 1865; died May 17th, 1866.

*Symptoms and History.*—Large tertiary ulceration on left shoulder; necrosis of frontal and parietal bones; diseased bone in nose; hard node on tibia; soft swelling on lower part of right fibula; old ulceration and partial destruction of soft palate.

The first symptom observed by this patient was the ulceration of the throat, which commenced a year and a half before her admission. After a few months there was evidence of disease in the cranial bones. The ulceration on the shoulder was of two months' duration. She had taken mercury on several occasions to salivation. This patient was in a very depressed and cachectic condition, and was much emaciated.

*Syphilisation.*—The inoculations were commenced on the chest by Dr. Böeck, September 8th, 1865, with matter from Case 1 (soft); this was unsuccessful. She was then inoculated twenty-nine times, and almost daily, with matter from various patients undergoing syphilisation, but the result was either complete failure or the production of abortive pustules, which were not reinoculable. At length on October 23rd a positive result was obtained on the right arm

with matter from Case 23 (soft), but it failed at the fourth generation. Matter was then used from Case 5, which ceased to take at the third generation. On the 13th November matter from Case 4 was used with a positive result, and went through a series of fourteen well-developed pustules. With the same matter at the same date inoculations were made on the thighs, and were carried through nineteen generations to January 13th, 1866. From this time she was inoculated regularly on the thighs with positive results, several prolonged series being obtained with matter from different sources up to April 28th.

There was no evidence of approaching immunity at this date (six months and twenty days from the commencement of treatment), when the inoculations were discontinued in consequence of sloughing of the cranial wounds having set in, with serious head symptoms. 468 inoculations were made; 254 of these were positive, 214 negative. The longest series derived from the same matter was nineteen (soft).

*Progress and Result.*—No improvement was observed during the first three weeks of the treatment, but the soft node on the fibula gave way and caused an ulcer. Iodide of Potassium with sarsaparilla was therefore ordered on September 29th, under which she improved in health and condition, and the inoculations, which had hitherto failed altogether, began to give more promise of positive results. In consequence of this improvement in the inoculations, Dr. Böeck discontinued the medicine on October 11th, but its omission was soon followed by an aggravation of the symptoms, especially by the rapid spreading of the ulceration over the fibula. The iodide was accordingly resumed on the 1st November and continued for three weeks; she then regained health and strength, and the ulcerations on the shoulder and leg began to heal. On the 22nd of November the iodide was again omitted.

From this time till February she continued to improve; the ulcers on the leg and shoulder healed almost entirely; she gained flesh and was able to walk about the wards. The inoculations were now uniformly successful.

In March her health began to fail and the ulcerations on the shoulder and leg again broke out; the iodide of potassium was therefore resumed,—this time, however, without any beneficial effect.

During April some loose portions of the parietal and frontal bones were removed, and early in May another piece of the parietal bone separated, exposing the dura mater. All this time the ulcerations were spreading, and she was seriously declining in health. Towards the end of April the inoculations were discontinued.

On the 10th May she was attacked with paralysis. The open wound at the back of the head had for a few days previously assumed a sloughing appearance. The sloughing extended to the dura mater, which gave way, and the brain protruded. Her paralytic symptoms increased, and she died May 17th, 1866. Other sloughing cases were in the wards at the same time.

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*Remarks.*—The remarks suggested by the cases above narrated may be divided into those relating to the value and effect of syphilisation as a method of treatment; and those having reference to the various pathological questions illustrated by the inoculations and their progress.

From the practical aspect of the question, the point to be first considered is the length of time occupied by the treatment. In those cases in which mercury had not been previously given, the inoculations occupied periods varying from six weeks to eight months and seven days. In the mercurial cases from three months and nineteen days to seven months and twenty days. The average in the first class was four months and three days; in the second class, five months and four days; and in the whole number of cases, four months and nine days.

This, however, does not represent the entire period during which the patients remained in the hospital, as the healing of the later inoculations required time, and was, in some of the cases, much protracted. Their stay in hospital averaged in



the non-mercurial cases, five months and fifteen days; in the mercurial cases, five months and nineteen days; and in the whole number, five months and sixteen days. The period is calculated from the date at which the inoculations were commenced.

The average duration of the sores produced by inoculation was about three weeks. The size of the ulcers varied in different individuals; their average diameter may be stated at about half an inch. They were as a rule smaller and, in every respect, milder when made on the trunk of the body than when made on the upper extremities; milder again on the upper extremities than on the lower. There was a general tendency towards diminution in size, and the sores progressed more rapidly as the treatment was proceeded with, till at length it was extremely difficult, and often impossible, to obtain any result from inoculation. When the susceptibility of one region of the body was exhausted (the trunk, for example), the inoculations would take freely on the arms, and when the arms had been subjected to a like process with a like result, they would still take freely on the thighs, till a period was reached when it was next to impossible to obtain pustules at all on any part of the body, or if obtainable, they were small and abortive, and the matter from them was not reinoculable.

The healing of the sores was in several of the cases delayed considerably beyond the average period mentioned, and this was observed to be the case more frequently on the lower extremities than elsewhere. The sores did not exhibit any great tendency to spread, the larger ones rarely exceeding the size of a shilling. Occasionally, however, where the punctures had been made too close together, larger sores were produced by two or more becoming confluent.

It is a very remarkable circumstance that in no case was there any perceptible affection of the neighbouring lymphatic glands resulting from the inoculations.

The pain occasioned by the inoculated sores was in some cases severe, in others it appeared to be trifling. It was in almost every instance cheerfully submitted to. It was found

to admit of alleviation to a great extent by careful dressing, so as to protect the sores from irritation and friction ; simple ointment was applied for this purpose.

The inoculations did not appear in any case to exercise a prejudicial influence on the health of the patients. On the contrary, most of them improved in appearance, and seemed to gain strength while the inoculations were going on. This, however, may have been partly owing to enforced regularity in diet and habits, and to improved hygienic influences. The pustules and their resulting ulcers were better developed in those patients who were in robust health, than in those who were weakly and out of health. The number of inoculations required, before the process was completed, varied greatly. The smallest number of pustules in any case was sixty-six (Case 14), the largest number was 296 (Case 5).

The patients are, of course, permanently marked by the cicatrices. The appearance of these is very similar to those occasioned by vaccination.

We will now refer to the results obtained. First—in those cases which were uncomplicated by any previous mercurial treatment, twenty-two in number.

In sixteen of these the treatment was persevered with until an immunity, more or less complete, had been arrived at, and by the time this was effected the syphilitic manifestations had disappeared. In these cases, therefore, the process may be considered to have been carried to its legitimate termination. This result was obtained—

In 1 case in	.	1 month 15 days.
1	„	2 „ 3 „
7 cases in from	.	3 to 4 months.
4	„	4 to 5 „
2	„	5 to 6 „
1	„	7 to 8 „

Of these sixteen cases, six (Cases 1, 3, 6, 10, 17, 18) have not been heard of since their discharge from the hospital, but ten have remained under observation up to the present time (April, 1867), or to within a very recent date. Of these ten,

eight (Cases 2, 5, 7, 8, 13, 14, 15, 16) have been inmates of the Lock Asylum; all of them have remained in perfect health and have had no recurrence of their disease. Of the remaining two, one (Case 11) returned to the hospital with a slight relapse, in the form of mucous tubercles on the genitals, which, however, soon disappeared under simple local treatment; the other (Case 9) has recently applied with swelling and superficial ulceration of the labium, but, as these rapidly subsided, and she presented no other syphilitic symptoms, we are disposed to think it was not connected with her former disease.

In the remaining six cases the treatment was not persevered with to the end. Four of them, viz., the three males (Cases 20, 21, 22) and one of the females (Case 19), left the hospital by their own desire before it was properly completed. Of these four, we have subsequent information respecting one only (Case 22), who, after having been inoculated for four months, returned with a relapse of his cutaneous eruption soon after the practice was discontinued.

In the two other cases, females, the treatment was given up before there was evidence of immunity, in consequence of sloughing having recurred in the cicatrices of former sores. One of these patients (Case 12) died from phagedæna. The sloughing, however, did not originate in, nor was it propagated to, any of the inoculated sores; it was confined to the labium, groin, and abdomen.

The other (Case 4) suffered in a similar way and to an alarming extent, but gradually recovered. In her, also, the sloughing was confined almost entirely to the genital organs, though one of the neighbouring inoculations was affected to a limited extent. This patient has recently presented herself at the hospital; she is now in sound health and in a situation as laundry-maid. She has had no return of her venereal symptoms.

It would not be fair, in our opinion, to attribute the untoward result of these two cases to the inoculation treatment. Both had been originally admitted with sloughing sores, after recovering from which, syphilisation was adopted

for their secondary disease. Nothing unfavorable occurred in one for three months, in the other for five months: but when immunity was nearly reached, sloughing commenced in the cicatrices of the old sores, and not in the inoculations. It would probably never have occurred, but for the presence of several cases of phagedæna in the wards at that particular time.

Next—as to the result of the treatment in those cases comprising Class 2, in whom mercury had been previously given; they were five in number.

Three of them (Cases 23, 24, 25) went through the process to its conclusion, in periods respectively of four months, five months, and five and a half months, and when discharged from the hospital were apparently free from disease. Of these three, one (Case 24) has not since been heard of, the other two have returned with relapses. These were, however, mild in character; one of the patients speedily recovered under bichloride of mercury, the other got well without any specific treatment.

The remaining two patients died. One (Case 26) from disease of the liver, supposed during life to be malignant, but found after death to be amyloid degeneration. She was inoculated persistently for more than three months with every kind of matter procurable, but with almost uniformly negative results. The cause of death in her case was therefore obviously quite unconnected with the treatment by inoculation. The other (Case 27) died from sloughing of the dura mater and exposure of the brain after the separation of a piece of necrosed bone, at a time when sloughing sores were prevalent in the wards. In our opinion the cause of her death also was in no way attributable to the inoculation treatment.

The results obtained in these five cases, then, were very unsatisfactory, but they corroborate Dr. Böeck's experience that the previous administration of mercury interferes not only with the progress of the inoculations, but with their therapeutical action. Out of three who went through it to the end, as many as two were known to have suffered from return of the disease. In the fourth, the inoculations having

been almost uniformly negative must be held to have gone for nothing one way or the other. In the fifth, it is by no means clear that the improvement which was at one time observed was due to the inoculations, rather than to the good diet and to the iodide of potassium, which she was taking at the same time.

With respect to the disappearance of the syphilitic symptoms while the treatment was going on, it was remarkable that in nearly all the cases no improvement took place for a considerable time—usually not until the second or third month—while in many of them the symptoms appeared to be aggravated, and fresh outbreaks of eruption were observed. Three patients were attacked with iritis (Cases 6, 15, and 18). In the two first the inflammation subsided under simple treatment with atropine and sedatives, and no injury to the eyes resulted. In the third the inflammation was more severe, and it was not considered safe to refrain from the use of mercury. This drug was accordingly administered in the usual way, and under its influence the iritis rapidly disappeared.

As an auxiliary to the treatment by syphilisation, nitrate of silver was frequently used to the mucous tubercles on the genital organs, and to the similar patches on the mouth or tonsils, as without local treatment the inoculations appeared to exercise only a slight and tardy effect upon them.

Having thus given a brief history of the cases treated, and a summary of the results obtained, the principal questions requiring an answer respecting syphilisation appear to us to be—

Whether the process exercises any material or appreciable influence over the evolution and progress of the disease; or whether the favorable results may not more reasonably be attributed to lapse of time, the regular habits and regime of hospital life, and the natural tendency towards recovery which the disease exhibits in persons otherwise healthy?

Whether—if it be admitted that syphilisation has a beneficial influence—that influence is of a specific character, such as Dr. Böeck has claimed for it; or may it rather be ascribed

as it has been by others, solely to the depurative and excretory action of the ulcers?

We do not pretend, from the limited number of cases which have been under our observation, to speak with authority on these difficult and important questions. Our object in this paper is rather to record facts, than to express opinions: but we would, nevertheless, with much deference, submit to the society the following conclusions as those at which we have ourselves arrived.

We will commence by saying that we are disposed to reject the hypothesis that the results are, in any appreciable degree, owing to a depuratory or excretory action of the pustules, analogous to what would be obtained by an equal amount of suppurative action artificially induced in any other way. Without denying that a discharge of pus from the cutaneous surface may have some effect in the elimination of a poison from the system, we think, if this explanation were correct, an amelioration in the symptoms would have been noticed at an earlier period of the treatment (as is the case, for instance, when mercury or iodide of potassium are given), whereas the rule has certainly been, either that the syphilitic manifestations remained stationary for a considerable time, or that they exhibited decided aggravations. And the depurative hypothesis is further disproved by what is observed in the ulcerations which so often accompany the chronic and tertiary forms of the disease; the suppuration from which has certainly anything but a curative action.

There remains then the question—has syphilisation any specific effect over the disease? or is it devoid of all appreciable influence whatever? It will be obvious how great is the difficulty of eliminating the element of time and the natural tendency towards recovery in the consideration of such a question; it will not therefore be thought surprising if our conclusions regarding it are not precisely in accord.

It is the impression of one of us (Mr. James Lane) from the observation of these cases, that syphilisation does exert some beneficial and specific influence over the progress of the disease, possibly in the way ascribed to it by Dr. Böeck. It

has appeared to him that the treatment conducts patients through the disease more safely and rapidly than if they are left to themselves ; that it leaves them with less liability to relapse, and that their relapses when they do occur are milder in character.

Mr. Gascoyen, on the other hand, thinks that the natural tendency to recovery which an early and uncomplicated constitutional syphilis exhibits with the lapse of time, and under circumstances favorable to the general health—such as the dietary, rest, regular hours, &c. of a hospital afford—is sufficient to account for the subsidence of the secondary symptoms during syphilisation ; and he is doubtful whether relapses are less frequent under this than under the ordinary methods of treatment. Neither does he admit any specific or other influence on the disease or the system from the continued inoculation of the virus. Were such the case, these fresh introductions of virulent matter would, in his opinion, either add to and intensify the existing disease, or else contaminate the blood until a general cachexia is produced, neither of which conditions is met with in practice, but the contrary. He, therefore, considers that no effect whatever is produced either upon the disease or the system by syphilisation.

Differing, however, as we do, on the scientific aspect of the question, we are entirely in accord as to its practical bearings, and we are decidedly of opinion that syphilisation is not a treatment which can be recommended for adoption. We consider that, even if it could be admitted to possess all the advantages claimed for it by its advocates, its superiority over other modes of treatment, or in many instances over no treatment at all, would not sufficiently compensate for its tediousness, its painfulness, and the life-long marking which it entails upon the patient.

If syphilisation could be relied upon, after other treatment had failed, to control the severer forms of the disease (especially in its tertiary stage), or to prevent its hereditary transmission, the benefit derived would, without doubt, more than counterbalance these disadvantages, but unfortunately

these are precisely the cases in which it has admittedly the least influence.

We will now refer to some points in the pathology of the disease which are illustrated by these experiments.

The matter employed for inoculation in these cases was obtained sometimes from hard, sometimes from soft sores. Whenever it was practicable, matter from hard sores was used. Dr. Böeck prefers this kind of matter for the purpose of syphilisation, having found it more rapid and effectual in its curative action, and less locally irritating; nevertheless he does not believe there is any essential difference in the operation of the two kinds of matter.

There has been a marked difference in the inoculability of the secretion from these two varieties of venereal ulcers. When matter from soft sores was used, pustules were readily obtained in the great majority of cases, *but not in all*. When matter from hard sores was employed, the same result was obtained, but with considerable difficulty and the failures were more frequent. The difference in the inoculability of the two kinds of sores would appear to depend in a great measure on the different character of their secretion; that from the soft sore consisting of well-developed pus, whilst that from the hard sore is scanty and serous, and often altogether destitute of pus-globules. This difference was, we believe, first pointed out by Mr. Henry Lee,<sup>1</sup> who also showed that if the hard sore were artificially irritated and made to secrete pus, it then became inoculable without much difficulty. This is in accordance with the experience of Dr. Böeck, who has observed that in proportion as the secretion is thin and serous, so will be the probability of failure. He therefore recommends that a piece of dry lint should be left in contact with the sore for twenty-four hours, at the end of which time there will commonly be found a thick purulent secretion; or this result may be more readily obtained by the artificial irritation of the sore with a stimulant, such as

<sup>1</sup> 'British and Foreign Medico-Chirurgical Review,' October, 1856, and April, 1859.



savine powder or savine ointment. From the matter thus formed a positive result can usually be produced by inoculation. In case of failure he inoculates daily, and in this way rarely fails of success.

The view that properly developed pus is necessary for the successful inoculation of syphilis (at all events on a syphilitic patient) is supported, *conversely*, by the experiment of Rollet, who found that if the secretion from a soft sore were deprived of its pus-globules by filtration, the remaining liquor puris was not inoculable.

Among the cases we have related will be found five examples of successful inoculation, made direct from typical indurated sores, carefully selected for the purpose, and as the possibility of inoculating a syphilitic patient from sores of this description has been denied of late years by many eminent authorities, we would direct special attention to these cases as tending to elucidate a much controverted question.

The first example was in Case 22, the male patient treated by Mr. Walter Coulson. This man had an indurated chancre on the prepuce and a secondary eruption. He was inoculated successfully from his own sore by Mr. Coulson, but it was not till after a daily inoculation for nearly three weeks that a positive result was obtained. From this a series of seven generations was produced, and the matter was used in the inoculation of several other patients.

The next instance was in a female (Case 9) who was successfully inoculated from the primary sore of the above male patient, after it had been irritated for two days with savine ointment. Six punctures were made, and all six were followed by a positive result. The pustules were well developed and were carried through a series of eleven generations.

A third example of direct inoculation from an indurated sore occurred two months afterwards in the same female. The matter was taken from a carefully selected indurated sore in a male out-patient. The sore having been irritated for two days with Unguentum Sabinæ, three punctures were made, two

of which failed, but one produced a well-developed pustule which was inoculated through nine generations.

A fourth will be met with in Case 7. The matter was taken from a well-marked indurated sore in a female out-patient, who afterwards had a secondary eruption. Six inoculations were made and all were successful. They were carried through ten generations. The sore, which was quite recent, was also inoculated successfully on the patient herself.<sup>1</sup>

A fifth will be found in Case 16, who was inoculated from a female patient with a well-marked indurated sore on the labium. In this case six punctures were made, but only one proved successful. It was reinoculable through twelve generations.

It appears then that there is greater difficulty in arriving at a positive result by inoculation with matter taken direct from a hard sore, than with matter from a soft sore, but when pustules are obtained, they are as freely reinoculable in the one case as in the other. They may be carried with equal facility through a lengthened series on the same patient, and transferred with equal readiness to others. In all these cases of inoculation from indurated sores, the pustules have been well developed, and have produced ulcers varying in size from a fourpenny piece to a shilling, but there has been nothing in their appearance to distinguish them from those made from non-indurated sores. There has been no more appearance of induration in the one than in the other; no noticeable difference in the duration of the ulcers (which averaged from three to four weeks), or in the appearance of the resulting cicatrices.

The instances we have mentioned afford, in our opinion, convincing proof that the hard sore may be inoculated on the

<sup>1</sup> This would furnish a sixth instance of successful inoculation from an indurated sore. A seventh example, which occurred at the Lock Hospital in February, 1866, may also be mentioned. The patient was a male, under Mr. W. Coulson, who succeeded after four consecutive days' inoculations, in obtaining well-marked, and reinoculable pustules. Both these are examples of auto-inoculation, and another is seen in Case 22.

bearer, or on another syphilitic patient. It appears to us impossible to explain them away, either by the theory of the mixed chancre, or by the supposition that they are all owing to an accidental contamination of the lancet with matter from soft sores.

But if the inoculability of the hard sore has been denied on insufficient grounds, these experiments go far to show that the inoculability of the soft sore has been much exaggerated.

It has been taught of late years that the distinctive character of the soft sore is its constant communicability by inoculation to all persons alike, and at all times, and whether they are the subjects of syphilis or not. This, according to our observation, is certainly not the case; it would appear that, sometimes from no obvious cause, but more often when there is great depression of the vital powers, the difficulty of obtaining a positive result by inoculating syphilitic matter is great, whether it be taken from hard or soft sores.

In one patient (Case 27) with severe tertiary syphilis, only an occasional, and then a very imperfect, result was obtained for more than six weeks, though she was inoculated almost daily; but at length as her health improved under the iodide of potassium and a liberal diet, pustules began to be developed. It was remarkable, also, in this case, that when the inoculations became successful, some of the earlier punctures, which had been apparently failures, came forward and produced pustules.

Another patient (Case 26), who was in a very depressed condition, and who ultimately died from disease of the liver, was inoculated over and over again with every variety of matter without effect. She was inoculated on forty-one different occasions, and in three or four only was anything approaching to a positive result obtained, and the pustules were then small and abortive.

In another female (Case 12), also in a very low condition, the inoculations at first failed, but succeeded afterwards and took freely, as her health improved.

In a fourth instance (Case 13) there was a very remark-

able temporary immunity during an attack of jaundice which lasted for about a month. In this patient, while she was jaundiced, no inoculations could be made to take, although they had done so freely before the affection commenced, and took again, with equal freedom, as soon as the jaundice got well.

In some other cases (5, 11, 14) there were also frequent failures, although the patients were apparently in sound health. It appears to us, therefore, from these numerous examples, that there are conditions, both natural and acquired, under which a person is exempt, temporarily at any rate, from the action of the virus even when obtained from a soft sore; and that some persons possess this immunity or power of resisting the effect of the poison much more than others.

In two cases (17 and 18) a circumstance was observed which is worthy of attention. When these two patients had been inoculated for more than four months, and when they had arrived at such a degree of immunity that it was impossible to obtain satisfactory pustules with the matter at our disposal, they were inoculated by the house-surgeon with matter from a spreading phagedænic sore, four punctures being made in each patient. In both cases sloughing sores were produced at each puncture. In Case 17 they coalesced, and spread to an alarming extent. In Case 18 they were less severe, though each puncture produced a sloughing sore of an inch and a half in diameter. These inoculations did not, even in their early stage, present the ordinary aspect of a syphilitic inoculation, but rapidly gave evidence of a spreading phagedænic action. It would seem, therefore, from these cases, that phagedæna may be communicated as such, directly, by inoculation, and this even in cases where there is an evident indisposition to inoculation with ordinary syphilitic matter.

The immunity to the action of the syphilitic virus obtained by repeated inoculation is another point of great interest. It appears to be beyond question that such matter cannot be

inoculated successfully *ad infinitum*, but that the susceptibility to its influence becomes gradually exhausted. At the commencement of the treatment the inoculations could, as a rule, be continued through a series of twelve or fifteen generations, without requiring the matter to be renewed, while the subsequent series became gradually shorter, until at length the result was altogether negative, or only small pustules were obtained, which could not again be inoculated. In many cases this immunity was tested repeatedly with matter from different sources. In some patients the immunity was less perfect, and with fresh matter two or three generations of pustules could be obtained, but these were small and imperfect, and rapidly healed, so that for all practical purposes the treatment came to an end. The longest series obtained in any case was thirty-eight (Case 5), and there were some between twenty and thirty. There were several exceptions to the rule that the first series was the longest, and the longest of all (thirty-eight) occurred towards the conclusion of the case.

In testing the degree of immunity, it should be remembered that the development of a small pustule, after the inoculation of new matter, is not sufficient evidence against the presence of this condition, as any irritant introduced beneath the skin may have the same effect. The only true test of a genuine syphilitic inoculation is, that it shall produce matter which is again inoculable.

It is also interesting to observe, that whether the matter employed was obtained from hard or from soft sores, the same degree of immunity was the result; and that the inoculation of matter from a hard sore would, in due course, cause insusceptibility to the action of that from a soft sore, and *vice versa*; and also when the patient had been treated partly with one matter and partly with the other, the immunity was equal against both.

*Data showing the duration of the treatment in each case and the number of inoculations made.*

The following numbers show the number of days in which each case was treated, and the number of inoculations made. (See column 1.)				Number of inoculations made.		Total.
Days.	Positive.	Negative.	Days.	Positive.	Negative.	
1	3	12	5	157	124	281
2	3	12	5	155	121	276
3	3	12	5	157	122	279
4	3	12	5	181	78	259
5	3	12	5	226	126	422
6	3	12	5	154	106	260
7	3	12	5	160	105	265
8	3	12	5	168	94	262
9	3	12	5	170	94	264
10	3	12	5	127	117	244
11	3	12	5	82	184	266
12	3	12	5	126	89	235
13	3	12	5	164	123	287
14	3	12	5	86	107	173
15	3	12	5	131	131	262
16	3	12	5	116	40	156
17	3	12	10	122	15	137
18	3	12	6	103	27	130
19	3	12	6	74	28	102
20	3	12	6	178	174	352
21	3	12	6	93	40	133
22	3	12	4	173	106	279
23	3	12	4	92	181	273
24	3	12	4	127	164	291
25	3	12	5	195	144	339
26	3	12	4	22	226	248
27	3	12	5	254	214	468
116	19	149	12	3913	3080	6993
Average 4	9	5	16	145	114	259

OBSERVATIONS  
ON THE  
TEMPERATURE AND THE URINE  
IN TYPHUS FEVER.

BY  
CHARLES E. SQUAREY, M.R.C.S.,  
RESIDENT MEDICAL OFFICER TO UNIVERSITY COLLEGE HOSPITAL; LATE  
RESIDENT MEDICAL OFFICER TO THE LONDON FEVER HOSPITAL.

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THE following observations were made whilst I was Resident Medical Officer to the London Fever Hospital. To Dr. Murchison my best thanks are due for allowing me to carry them out on patients admitted under his charge, and for many valuable hints in connection with them.

Observations were made on eighteen cases.

The temperature in each case was taken four times daily, as punctually as possible at the stated hours whilst it was above the normal point, and afterwards twice daily till the patient left the Hospital.

The urine was collected every morning at 8 a.m., and examined the same day for urea and chlorides.

The analyses were made according to Liebig's volumetric method. In analysing the urea the chlorides were not separated, but allowed for in proportion to the quantity present.

The colour of the urine has also been carefully noted, and in doing so the urine was daily compared with the table of colours, given in Neubauer's and Vogel's book 'On the Urine.'

A full account of each case with the temperature chart and daily analysis of the urine will be found at the end of this paper. I will here merely state that, in all the cases, the date of the invasion of the disease was accurately ascertained, that in almost every case it was characteristically sudden, and that in all it was well marked by rigors, headache, and general pains.

That of the eighteen cases the first ten were very bad, being accompanied with much delirium and stupor, and having copious dark petechial rashes, with dry brown tongues and sordes-covered teeth and lips. In all these there were the peculiar vacant "typhus" expression.

Of these cases one died.

Of the eight others, Case 15 was the worst. In each, with one exception, there was a distinct though not very copious rash. The tongues remained moist throughout, or were only dry for a day or so during the attack. The delirium and stupor were less, the patients could be more easily roused and could be made to answer questions, though certainly not truly with regard to their condition.

In one case, No. 18, there was not at any time a distinct rash, only slight mottling being noted for a day or so after admission. The boy, however, had come from a house where typhus fever was rife, his father and brother had been in the hospital with the disease, and from there being no other symptoms to account for his high temperature, which ran much the same course as typhus fever although of shorter duration, I think it not unfair to conclude that it was a mild attack of this disease. During the time that he was in the hospital he was in a typhus fever ward, and the fact of his not contracting it also points to the same conclusion.

One case, No. 15, was admitted on the second day of the disease, and on the third day, with regard to the rash, it is noted that the skin of trunk is injected with, here and there,



some slightly elevated rose-coloured spots which disappear entirely on pressure. On the fourth day the typhus rash was distinct.

Case 9 was admitted on the third day; in this case the rash did not appear till the fifth day of the disease.

*Complications noted.*

*Diarrhœa* was present more or less in seven cases (Nos. 1, 2, 5, 8, 9, 15, and 16). In all but Case 1 it was easily stopped by a mixture containing chalk and catechu. It was not accompanied in either case by any tympanitis or tenderness of the abdomen.

*Respiratory organs.*—*Bronchitis* was present more or less in every case, excepting Nos. 11, 17, and 18. In Cases 1, 2, and 3 there was much congestion of the bases of the lungs as well. Case 1 died, and in Case 2 consolidation of both bases followed the congestion, and this again was followed by well-marked evidence of gangrene of a circumscribed portion of the lower lobe of the left lung.

*Heart.*—The impulse could scarcely be felt, and the first sound was noted to be feeble in all cases during the second week of the attack.

In Case 15 a soft blowing systolic murmur of maximum intensity at the left apex, scarcely audible at base, was noted from the twelfth to the nineteenth days, not afterwards.

Reduplication of the second sound was audible more or less in every case.

In Case 6 convalescence was retarded by occasional boils and sore throat, causing sudden and very high elevations of temperature.

*Treatment.*—This has been in most cases simply Mist. Camphoræ ʒj, every four hours, followed by a mixture containing either bark or quinine as soon as convalescence was established.

Cases 3, 4, 7, and 13, took a mixture containing ether and sulphuric acid instead of Mist. Camphoræ; and Cases 15 and

16 a mixture containing the dilute nitric and hydrochloric acid.

Brandy was given in Cases 1, 2, 3, 7, 11, and 12; porter or wine in almost every case during convalescence.

The diet consisted of milk and beef-tea with arrowroot and eggs during the acute stage, followed by fish or meat as soon as patient felt able to eat.

*The temperature.*—The range of temperature in the following cases agrees very closely with the general remarks made by Dr. Buchanan, in his paper 'On Typhus Fever,' in Reynolds' system of medicine.

1st. These cases show that the rise of the temperature at the commencement of the disease is very rapid.

2nd. That the range of temperature the first week or so continues very high, and that it varies very slightly.

3rd. That a well-marked remission in the temperature generally occurs between the seventh and tenth days of the disease.

4th. That the lysis is gradual and commences in uncomplicated cases with this remission.

5th. That the lysis may be interrupted by exacerbations which may be produced either by complications or by some external disturbing influence.

6th. That during the first week of convalescence the temperature is rather low, rising again after a variable period, depending on the severity of the attack, and the constitution of the patient, to the normal line or even higher.

7th. Four of these cases show that during convalescence the temperature may rise to above 100° Fahr. (37·7° cent.), and continue so for a day or two without there being any assignable cause.

The following are the facts on which each of these statements is founded :

1st. The rapid rise of temperature at the commencement of the disease is shown by Cases 9 and 15, in which it rose to 105° Fahr. (40·5 cent.) and 104·6° Fahr. (40·3° cent.) respectively on the third day of the attack.

In Case 18 a temperature of 104·6° Fahr. (40·3° cent.) was

noted on the second day, but in this case there was no distinct typhus rash.

2nd. The high and slightly varying range of temperature during the first week or so is shown by thirteen cases, in all of which the temperature remained above  $103^{\circ}$  Fahr. ( $39.4^{\circ}$  cent.) during this period.

Six of these cases were, though only admitted on the seventh day of the disease, so that in these the foundation for the above rests only on one or two observations taken on the day of admission, when it might be that the excitement and disturbance consequent on the removal of the patient to the hospital had caused the temperature to rise higher than it otherwise would have done. Still, of the seven other cases admitted, two on the third, three on the fifth, and two on the sixth day, the temperature continued above  $103^{\circ}$  Fahr. ( $39.4^{\circ}$  cent.), till the seventh day, and there was no particular difference noted in either of them between the temperature, the day of admission, and that of the succeeding day.

The slight daily variation that existed at this time, or rather before the commencement of the lysis, is shown by fourteen cases in which it was under  $1.5^{\circ}$  Fahr; the difference between the night and morning temperature of succeeding days was in eight cases  $1^{\circ}$  or less, in six cases  $2^{\circ}$  or less.

The highest temperature noted  $105.2^{\circ}$  Fahr. ( $40.6^{\circ}$  cent., in two cases.

The maximum point was reached—

In one case (No. 18) on the second day; in four cases on the fifth and sixth days; in three cases on the seventh day; in four cases on the eighth day, in one case on the tenth day; and in one case on the eleventh day.

The five cases in which the maximum temperature was noted on the eighth and tenth days did not, however, come under observation till the seventh day.

3rd. That a well-marked remission in the temperature generally occurs between the ninth and tenth days is shown by all the cases. In Cases 1, 4, 15, and 17, it was very slight. In Case 18 it occurred on the fifth day.

4th. That the lysis is gradual and commences in uncom-

plicated cases with this remission is shown by eight cases (Nos. 2, 3, 4, 7, 12, 13, 15 and 18). In three of these cases it occupied three days, in the five others a still longer time.

Thus it is seen that these observations do not confirm the statements made by Dr. Buchanan and Aitken, both of whom say that "the abrupt manner in which the fever leaves the patient is peculiar to the disease, that the lysis is frequently completed in twenty-four hours, sometimes in less.

The observations of Dr. G. S. Smith, of the Edinburgh Hospital for Sick Children,<sup>1</sup> and those of Dr. Perry, of Glasgow, and Dr. Compton, of Dublin, also coincide with these observations as to the more gradual decrease of the lysis. And Dr. Gairdner, as far back as January, 1865, states of the crisis, "that it is rarely quite rapid or sudden, usually extending over two or three days, and often rarely appreciable till it has been forty-eight hours or more in progress."

Generally the lysis commences with slight daily remissions. The daily fluctuations become also greater, and these increase the nearer the temperature approaches the normal point, as in Case 3. It may, however, be quite gradual throughout, as in Case 7, and in some, as in Case 12, it will be seen that the daily remissions at the commencement of the lysis may be as great as those at its termination.

The duration of the lysis depends on the severity of the attack and on the constitution of the patient, in severe cases being prolonged to the end of the third instead of the end of the second week.

In these cases complete defervescence was established, on or before the fourteenth day in eight, before the eighteenth day in the others.

5. In nine cases a slight exacerbation of the temperature has occurred after the commencement of the lysis, lasting for a day or more. In eight of these no distinct cause could be discovered for its occurrence. In all the patients were more delirious at this time. In Case 9 the rise in the temperature was due to the disturbance and noise made by a wildly delirious case in the same ward.

<sup>1</sup> See 'Edinburgh Medical Journal,' March, 1866.

6. That during the commencement of convalescence the temperature is lower than normal, is shown by four cases, in which a temperature of 96·8° Fahr. (36° cent.) was noted about this time.

7. In four cases during convalescence the temperature is seen to have been much above the normal point. In neither of these cases could any complication be found to account for it, and although in Case 15 the temperature was above 101° Fahr. (38·3° cent.), on the twentieth day, his appetite was very good, and he expressed himself as feeling very well.

*Diagnosis and Prognosis.*—Of the following cases there are only two, Cases 9 and 15, coming under observation before the characteristic rash appeared, from which any conclusions can be drawn as to the value of the thermometer as a means of diagnosis between the acute specific fevers.

In both these cases the temperature was above 104° Fahr. (40° cent.) on the third day of the disease, but such a temperature will not, as far as I know, exclude typhoid fever on the one hand or smallpox on the other.

In scarlet fever the appearance of the rash on the second day and in measles the special premonitory symptoms will enable us to distinguish them sufficiently early ; but in smallpox and especially varioloid, typhus fever, and typhoid fever, very little help, unless the case be well marked, is afforded by the early symptoms.

The rapid rise and the continued elevation of the temperature in these cases foretell the invasion of a serious disease, but I do not think it enables us to foretell, with certainty, which one of these fevers is approaching.

With regard to *prognosis*, the temperature during the second week will generally give great help. A very high temperature during the first week is not of so much importance as continued elevation in the second week. And of more importance still than this continued elevation is an "abnormal temperature," that is, the temperature falling rapidly, whilst all the other symptoms continue with unabated severity, see Cases 1 and 2 ; and also a case, L. R—, one of three cases published in the 'Lancet' of December 9th, 1865,

Dr. Compton, of Dublin, has also noted "that an abnormal course of temperature much more generally precedes a fatal termination than any unusually high range."

A too favorable prognosis should not be given from the remission that occurs between the seventh and tenth days of the disease, for although it indicates that the disease is running a favorable course, it does not denote that the most dangerous period of the disease is passed.

The temperature and the pulse, although corresponding in their fluctuations more or less in every case, have not done so so regularly that any number of beats can be said to correspond with any particular amount of variation in the temperature.

Out of these eighteen cases the following irregularities, so to say, have been noticed. Thus, in one case (No. 2), the temperature has risen at a much greater rate than the pulse, the former being 103° Fahr. (39·4° cent.), and the latter only 92 on the seventh day ; the pulse was 120 on the twelfth day.

In two cases the temperature being very high, the pulse remained low throughout. Thus, in Case 5, the temperature being 104° Fahr. (40° cent.), the pulse remained below 100, though the range for the last week of the patient's stay in the hospital, when he was feeling well and strong, was between 70 and 80 beats per minute, and in Case 11 it was much the same.

In three Cases (Nos. 8, 12, and 18) the temperature reached the normal rate before the pulse.

In seven cases the pulse was normal before the temperature, and this is most marked in Cases 13, 14, 16, and 17.

### *The Urine.*

There are at present very few published observations on the urine in typhus fever.

Besides the one case by Dr. Parkes', I know of only two others by Mr. Duunett Spauton in the 'Medical Times and Gazette' of 1864, and of a series of six cases by Dr. Keith

Anderson, published in the 'Edinburgh Medical Journal' of February, 1866.

Dr. Anderson's observations differ entirely from those of Dr. Parkes, the result that Dr. Anderson arrives at being that "the quantity of urea excreted daily during the second week of the disease is decidedly below the standard of health, notwithstanding that the patients were in a high state of fever, with a temperature and pulse much above the normal rate." He does not from this infer that there is a lessened formation of urea, but that it is not eliminated at this period, but retained in the system to be discharged later in the disease, yet neither of his cases, so far as they are published, show that the great retention of urea which must have occurred in every case is at all made up for by increased elimination at any time afterwards. There is also, in his observation, a great discrepancy to be noted in every case between the proportion of urea and the total solids as calculated approximately from the specific gravity. Take, for instance, the twelfth day of Case 1, where the urea amounts to only 18·05 grains in the whole twenty-four hours. The total solids are that day 354·7 grains, or nearly twenty times as much as the urea. The chlorides being very much diminished and the sulphates only slightly increased (Parkes), of what can these other solids, supposing the urea to have been rightly determined, be made up of? I have taken an extreme instance, but still this discrepancy exists more or less in every case.

Dr. Anderson's analyses were made according to Davy's method; and without in the least wishing to undervalue this mode of analysis, I would merely point out that, of all, it is the one to give the most unsatisfactory results unless the greatest care is taken in its performance.

Dr. Buchanan, from the results of his own experiments, states that the daily quantity of urea during the first week, when the patient is fed on low diet, milk and beef-tea, is about double that of the fourth week, when he is sitting up and eating his fill of meat. That the increase has been found at the earliest day at which the urine has been examined.

The analyses in the following cases were made according to

Liebig's volumetric method. In analysing the urea, the chlorides were not separated, but allowed for in the following ratio in every case.

1. When the chlorides for the whole twenty-four hours were less than 1 gramme, nothing was allowed for them.

2. When the chlorides were less than 5 grammes during the whole twenty-four hours, 1 cubic centimètre was deducted from the mercury solution used before calculating the urea.

3. When the chlorides were more than 5 grammes for the whole twenty-four hours, 2 cubic centimètres were deducted, as is generally allowed.

Albumen when present was separated before determining the urea.

The weight of each patient has been noted once or twice in the course of the disease. In calculating the amount of urea and chlorides excreted per kilogramme of body weight, the patient's weight on leaving the hospital has always been taken; and again, in making these calculations, I have not taken any stated time such as first and second weeks of the disease, but have in each case divided the attack into the following periods:

1st. That before the commencement of the lysis.

2nd. That occupied by the lysis itself.

And 3rdly. The remaining time of patients' stay in hospital has been divided into first and second weeks of convalescence, &c., as I think that by these means only can it be seen whether any relation exists between the excretion of urea and the temperature.

The following is a short account of the urea in each case.

CASE 1.—William L—, æt. 29, weight not known. In this case only one day's urine could be obtained before the patient's death, as he was very delirious, and suffering much from diarrhœa. He passed on this seventh day 910 c.c. S.G. 1025 acid, and it contained a little albumen; the urea was 37·310 grammes, the chlorides 1·820 grammes. It was a reddish yellow colour and had a copious deposit of lithates; as the disease advanced the albumen became very copious.



**CASE 2.**—Henry W—, æt. 51. In this case, before the commencement of the lysis, no urine could be obtained. During the lysis and the first week of convalescence he was passing more than the normal quantity, it being per kilogramme of body weight  $\cdot 697$  and  $\cdot 677$  of a gramme respectively.

During the second, third, fourth, fifth, sixth, and seventh weeks of convalescence it was much below the normal amount, especially in the fourth week, when it was only  $\cdot 328$  of a gramme per kilogramme of body weight. During the eighth and ninth weeks of convalescence he passed the normal amount. The patient, during his stay in the hospital, was in a very low weak state, suffering much from cough and expectorating a quantity of gangrenous smelling sputa. When he left he was still thin and weak, his cough was better and his sputa scarcely at all foetid.

Of the remaining cases, in 6 (Nos. 3, 4, 7, 12, 15, and 16) the urea excreted was above the normal rate both before and during the lysis, and in Cases 7 and 15 remained so even during the first week of convalescence, falling afterwards to less than the normal rate.

In four cases (Nos. 8, 9, 10, and 18) the urea excreted was much above the normal rate before the lysis, but less than normal during the lysis and for a variable time afterwards.

In five cases (Nos. 5, 6, 11, 14, and 17) the urea excreted was less than normal before the lysis, above the normal rate during the lysis, and in Cases 11 and 14 it remained high during the first week of convalescence.

In Case 13 no urine was obtained before the commencement of the lysis; during the lysis the urea excreted was above the normal rate, less afterwards.

In the four cases Nos. 8, 9, 10, and 18, the excretion of urea per kilogramme of body weight before and during the lysis was as follows :—

				Gramme.	The normal being
In Case 8, æt. 18, before the lysis	...	...	...	·616	} ·500 of a grain.
during „	...	...	...	·497	
„ 9, æt. 13, before	„	...	...	·748	} ·600 „
during „	...	...	...	·571	
„ 10, æt. 17, before	„	...	...	·658	} ·500 „
during „	...	...	...	·400	
„ 18, æt. 11, before	„	...	...	1·170	} ·800 „
during „	...	...	...	·671	

So that only in Case 10 is the urea much below the normal amount during this period (the lysis), in the others very slightly so, and considering that for some time the patients had been in bed living on low diet (milk and beef-tea), it must be allowed that although below the normal rate, it was still a large quantity for such patients to pass.

Of the five cases (Nos. 5, 6, 11, 14 and 17), in which the excretion of urea was below the normal amount before the lysis, whilst the patient was in a high state of fever, it will be seen that in one case (No. 5) the patient was at this time suffering from severe diarrhœa, which would perhaps be sufficient to account for it; that in this case, and also in Case 6, the urea was decidedly above the normal rate during the lysis, and that besides in both these cases only one day's urine was obtained at this period, and it will be allowed that the urine of one day is not sufficient to found an average on, in a disease where the daily excretion may be so irregular.

That in two more of the cases (Nos. 11 and 14), in each of which two days' urine were obtained at this time, the urea is seen to be only slightly below the normal amount, it being per kilogramme of body weight ·485 and ·473 of a gramme respectively, whilst during the lysis and first week of convalescence, especially in Case 14, the urea is so largely increased that it points rather to the lessened quantity of urea before the commencement of the lysis to be due to its non-elimination rather than its non-formation.

That in the other case (No. 17) six days' urine were obtained before the commencement of the lysis. During this time the excretion of urea was very regular, and the amount passed was much below the normal quantity, it being per

kilogramme of body weight only .438 of a gramme, the normal being about .600. During the lysis it was decidedly above the normal rate, but as the lysis only occupied three days, and the excretion of urea during this period was not sufficient to make up for the diminished excretion of the previous days, I think in this case it must be allowed that during the high temperature before the commencement of the lysis, there was not only a diminished elimination of urea but a lessened formation as well.

The daily excretion of urea in these cases has not been so regular as in that one of Dr. Parkes'.

In his, during ten febrile days, the range was one gramme below the mean of the ten days, and one and a half grammes above it.

The least variation in the daily excretion of urea that I have noted was in Case 3, the range being for four febrile days 5.177 grammes above and 3.915 grammes below the mean amount.

The largest amount of urea excreted on any one day was 65.570 grammes by Case 15 on the twelfth day of his attack, the temperature the previous day having been above 104° Fahr.

Patient's age was twenty, his weight 52.3 kilogrammes.

From these analyses the following conclusions are drawn :

1st. That during the first period of an attack of typhus fever before the commencement of the lysis, there is an increased formation, and generally an increased elimination of urea.

2nd. That during the lysis, and sometimes during the first week of convalescence, the *urea* is generally excreted at more than the normal rate.

And 3rd. That for a variable period during the commencement of convalescence the urea is much diminished in quantity, rising again to the normal amount as the patient regains his health and strength.

*The chlorides.*—The analyses were made according to Liebig's volumetric method.

When albumen was present it was separated by the pro-

cess described in Neubauer and Vogel's book on the urine, before the chlorides were determined.

Of the eighteen cases, Case 1 died; before his death only one day's urine was obtained. On this the seventh day, the chlorides were much below the limit of health, being only 1.820 grammes.

In the other seventeen cases the chlorides were excreted at much less than the normal rate during the pyrexial period and for a variable time afterwards; thus—

In five cases Nos. 5, 6, 11, 12, and 17) the daily average of chlorides was less than one gramme both before and during the lysis, gradually increasing after this time.

In three cases Nos. 4, 9, and 14, it was less than one gramme before the commencement of the lysis.

In two cases 2 and 16 the daily average of chlorides was less than one gramme during the lysis; and in the seven other cases the daily average was more than one gramme throughout the pyrexial period, though much below the normal rate.

The first increase noted in the excretion of the chlorides has occurred in eight cases before the temperature has reached the normal point; in two cases just at the end of the lysis; and in the remaining seven cases two or three days after the normal point had been reached.

This increase has in all cases corresponded with the return of the appetite, and the large quantity of chlorides daily excreted in some of these cases during convalescence, as in Cases 2, 5, 10, 13, 14 and 17, where it is seen that the excretion is much above the normal quantity, is most likely due chiefly to the large quantities of food taken at this time by the patients.

Four cases left the hospital before the excretion of chlorides had reached the normal rate, but in the remaining thirteen the excretion was much above the normal quantity when they were discharged.

The average daily excretion of chlorides has remained below the normal rate in each of these cases for a variable period.

Thus in one case, No. 4, only till the end of the lysis, the excretion then so rapidly increasing, that during the first week of convalescence, the daily average was above the normal rate.

In eight cases (Nos. 5, 7, 9, 10, 13, 14, 16, and 17) the average daily excretion was below the normal quantity till the end of the first week of convalescence.

In five cases (Nos. 6, 11, 12, 15, and 18) it was below the normal quantity till the end of the second week of convalescence.

In two cases (Nos. 8 and 3) till the end of the third week of convalescence.

And in one case (No. 2) till the end of the fourth week of convalescence; in this case, though, there was a lung complication.

In all these cases more or less salt was taken with the nourishment, whilst the patients were on low diet, though, no doubt, much less than is generally taken in health.

These cases thus prove what Dr. Parkes states, namely, that the chlorides are almost entirely absent during the pyrexial period. Their absence seems to be due *chiefly* to the lessened ingestion of salt in the food at this time. Their absence, though, cannot be entirely explained by lessened ingestion, as Dr. Murchison quotes two cases where salt was given in large quantities during the height of the fever, and yet was not discovered in the urine, although it was examined carefully for them for several days after.

In these cases the chlorides must either have been retained in the system or not absorbed at all.

In my own cases I think the large increase in the elimination during convalescence points to a certain amount of retention having occurred during the pyrexial period.

#### *The amount of urinary water.*

As nothing whatever is known in these cases with regard to the ingestion of fluid, which has so much influence on this

secretion, the following statements must be taken in a very general way.

I will only here say that the patients were not at all stinted in their drink at any time of the disease, being allowed plenty of water as well as their fluid nourishment during the twenty-four hours.

The mean of a number of experiments on the amount of this excretion during health collected by Dr. Parkes is 1500 cubic centimètres. This I propose to consider as the normal amount, and to compare with it the amount of urinary water in these cases during each period into which they have been divided. Thus—

1st. *Before the lysis.*—In only one case has the average daily excretion of water during this period been above the normal rate (1500 cubic cent.). In the sixteen others it was below this, and in seven of them below 1000 cubic cent.

2nd. *During the lysis.*—In four cases the amount of water was above the normal rate. In thirteen others below, and in five of these below 1000 cubic cent.

3rd. *During the first week of convalescence.*—The amount of water was below the normal rate in all cases below 1000 cubic cent. in eight.

4th. *During the second week of convalescence.*—Out of fifteen cases, in five the amount of water was above, in ten below the normal rate.

5th. *During the third week of convalescence.*—Out of nine cases it was above the normal quantity in four cases, below in five cases.

6th. *During the fourth week of convalescence.*—Out of five cases it was above normal in four, below in one case.

Therefore from these observations I think we may state in a general way that the amount of water excreted by the kidneys is diminished both before and during the lysis, and for a variable time afterwards, but that, about the end of the third week of convalescence, the quantity passed has reached the normal rate or more.

*Colour of the Urine.*

This has been determined by the plate of colours given by Vogel. He distinguishes three groups—the yellow, the reddish, and the brown or dark group.

In the one case that died the urine was for a day or so before a brownish-red colour; it contained a quantity of albumen and, judging from the colour, blood.

In the other cases the colour of the urine has for the most part belonged to those of the second group. Reddish-yellow and yellowish-red being the predominating colours during the pyrexial period and for some time after, then gradually losing the reddish tint and toning down into the first group; in some cases becoming almost colourless. There has generally been a copious deposit of lithates during or towards the termination of the pyrexial period.

*Albumen* was noted in twelve out of the eighteen cases at one time or other of the disease. In neither case was the urine examined microscopically for casts.

In five cases the urine was found alkaline for a day or so during the disease, and generally about the end of the third week.

*The Temperature Chart.*

Each large square represents one day's observation, the smaller blue squares show the division of each degree into fifths. The vertical column on the left side of the chart gives the temperature in the Fahrenheit scale, that on the right the temperature in corresponding degrees of the Centigrade scale. The three horizontal columns at the top of the chart indicate the day of the disease, the hour at which the observation was made, and the pulse.

CASE 1.—William L—, æt. 29. Admitted into the London Fever Hospital, March 26th, 1866. Is a labourer by

trade ; married. Says that he has been ill four days. His attack commenced with headache, chilliness, and general pains. On admission his mind was quite clear ; his skin was hot, and there was a distinct typhus rash on the forearms and back of his hands, less on his trunk. His tongue was moist and thinly furred, his bowels had not been open for a day or so. He had no cough, but he complained of much headache and general pains.

*Rash.*—Was well out on his trunk and limbs on the sixth day ; there was very little mottling, and the spots were large, rather dark, and irregular in size. It grew very dark and petechial, but faded somewhat before his death on the seventeenth day.

*Sleep.*—Bad from the commencement. Wandered, and was very restless from the seventh to the ninth, then became heavy and drowsy, with only occasional low muttering delirium. Patient could answer questions truly till the tenth day, then grew confused, and after the thirteenth he did not notice anything, lying in a heavy stupid condition.

*Complexion.*—Muddy, slightly flushed at first, but pallid after the tenth day. His pupils were small, the conjunctivæ suffused and injected. Lips dry, with sordes after the eleventh day. Expression dull and vacant after eighth day.

*Tongue.*—Dry and rough on the ninth, was brown on the thirteenth day, and caked over with dark-brown fur on the sixteenth day.

*Bowels.*—Confined at first, were loose more or less after the tenth day. Urine was passed in bed after the ninth day, and also feces after the twelfth day.

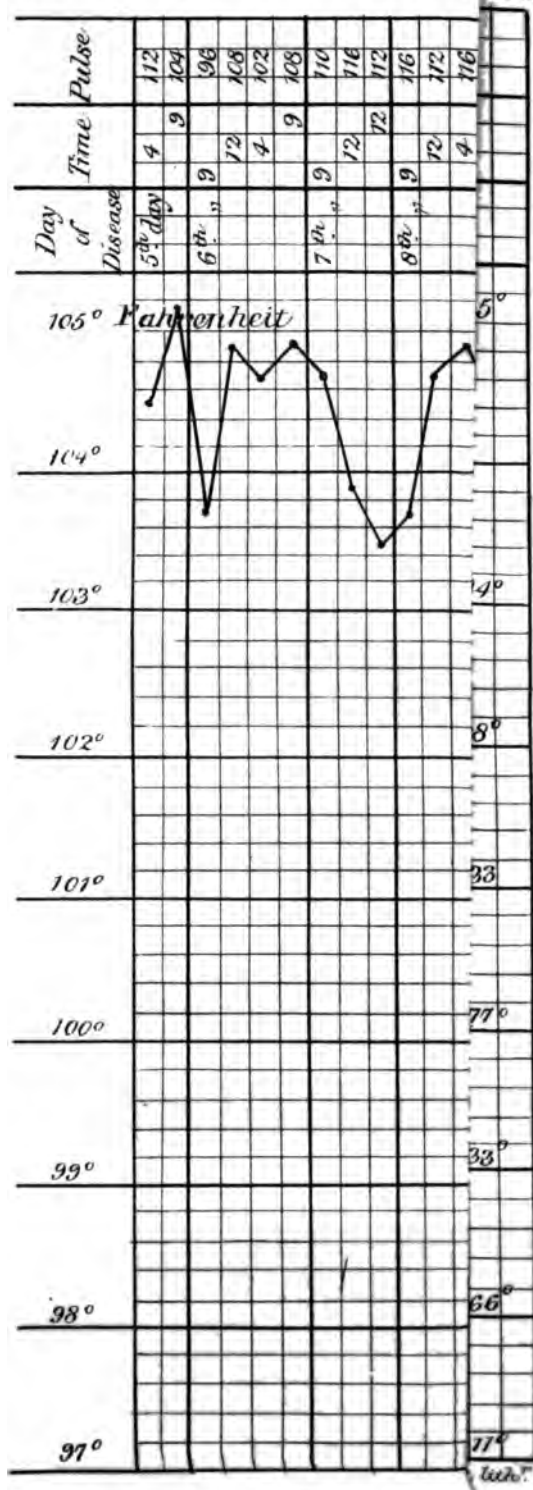
*Thirst.*—Complained of at first. Took drinks very badly on the fifteenth and afterwards.

*Perspiration.*—Copious on the nights of ninth and tenth.

*Cough.*—Patient had some troublesome cough on admission, with slight expectoration. On the eighth day some bronchitic râles were audible over the back ; these increased, and with them at bases towards end of disease some large moist râles were audible. There was some dulness on percussion at bases of lungs on the twelfth day.







*Heart.*—The pulse was very weak on admission. First sound was inaudible at base after the eleventh day; both were audible at the apex, but much muffled in character.

In this case only the urine of the seventh day was saved, the patient in his delirium passing it in bed. He passed on this day 910 c.c., S.G. 1025; and containing a trace of albumen.

The treatment was, in this case, large quantities of brandy daily, 12 oz. daily. Counter-irritants to the back and chest, and various remedies were tried to stop the diarrhœa. (See Diagram I.)

CASE 2.—Henry W—, æt. 51, admitted into the London Fever Hospital, November 7th, 1865. Has been ill six days. Is married, and a labourer by trade. Says that he is very poor indeed, and has not been living at all well. He says his present illness commenced six days ago with headache, shivering, and general pains.

On admission his skin was hot, and there was a distinct rather petechial typhus rash on the trunk and limbs. His tongue was moist and covered with a white fur; his bowels had been open the day before admission. He complained of little headache and some slight cough. His lungs were clear at bases.

*Rash.*—Grew very dark and petechial as the disease advanced. It began to fade on the fifteenth day, but slight remains of it were visible till the twenty-fifth day.

*Sleep.*—Till eleventh day pretty good, with little wandering. From eleventh to the sixteenth day there was much low muttering delirium, and patient was very restless, scarcely sleeping at all. After this he slept somewhat better, only wandering slightly every now and then. Between the twentieth and thirtieth day his cough became very troublesome and prevented his sleeping at night.

*Complexion.*—Sallow, with suffused and slightly injected eyes, the pupils rather contracted, and sordes on the legs and teeth from the eleventh till the eighteenth day. Then

slightly improved, but remained pallid and sallow till he left the Hospital.

*Tongue*.—Moist and furred till eleventh day, then became dry and brown, and continued so more or less till the nineteenth day, then gradually improved, but remained dry down the centre for some time, and after this became red and swollen.

*Bowels*.—Rather loose on the eleventh day; were confined after this, not being open without medicine.

*Headache* and general pains not complained of after admission.

*Appetite*.—Began to return about the twenty-eighth day but for a long time after this was very variable, sometimes pretty good, at others entirely gone.

*Thirst*.—Complained of slightly the first few days, but not afterwards.

*Mind*.—Patient remained in a very low stupid confused state for a long time after the fever had left him.

*Lungs*.—During the pyrexial period he had much bronchitis and congestion of the lungs, but not much dulness on percussion at bases. About the twenty-sixth day he complained of pain in the left side on drawing a deep breath, and on coughing. His cough became more troublesome, and he expectorated more; on the twenty-eighth day there was dulness on percussion at the left base, and round left side, with fine crepitation audible on deep inspiration, almost as high as the angle of the scapula. On the thirty-eighth day this dulness was noted as a circumscribed patch at the left base, about three inches in diameter, with some tubular breathing, and coarse crackling audible over this region. The expectoration was at this time a dark greenish colour, and very foetid. His breath was also very foetid. On the sixty-eighth day the following note was taken:—"On looking at back the left side is seen to be much flattened, a regular depression exists from about half way down scapular nearly to base, left side expands very little during respiration. No râles are here audible, and the respiration is bronchial in character. The expectoration is not so copious, but still very foetid. Patient



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left the Hospital a few days after this very weak, but still gradually improving.

*Heart* sounds were very weak indeed throughout.

Treatment consisted in this case of the æther mixture at first, and then stimulants and tonics. At one time, about fortieth day, he was taking a mixture containing free chlorine. Counter-irritants were frequently applied to his back, and linseed poultices almost continually.

During the height of the fever, he was taking 12 oz. of brandy daily; and afterwards, till he left the Hospital, about 6 oz. a day, besides beer. (See Diagram II.)

CASE 2.—Henry W—, æt. 51. *Analysis of Urine.*

Day of disease.	Quantity of water.	Specific gravity.	Reaction.	Albumen.	Urea.	Chlorides.		Colour and deposit.
	C. C.				Grammes.	Grammes.		
9	1000	1015	Acid	None	21.500		During lysis.	Reddish-yellow colour; deposit of lithates.
10	680	1020	"	"	20.060	.680		Yellowish red; cloudy deposit.
11	800	1019	"	"	23.600	.600		"
12	740	1018	"	"	19.240	.370		"
13	Lost	1018	"	Trace	.....	.....		Reddish yellow; deposit of lithates.
14	"	1015	"	None	.....	.....		"
15	"	1017	"	"	31.115	.685		" no deposit.
16	1270	1016	"	Trace	.....	.....		"
17	Lost	1013	"	"	31.860	.590		Yellowish red; cloudy deposit.
18	1180	1017	"	"	32.640	1.580		"
19	1020	1017	"	"	26.500	2.000		" deposit of lithates.
20	800	1020	"	"	26.650	2.460		"
21	820	1021	"	"	21.450	2.970		"
22	660	1021	"	None	.....	.....		Reddish yellow; copious deposit of lithates.
23	Lost	1026	"	"	18.525	2.565		Yellowish red
24	570	1022	"	"	17.400	3.190	1st week of convalescence.	" cloudy deposit.
25	580	1024	"	"	.....	.....		"
26	Lost	1026	"	"	19.100	5.440	2nd week of convalescence.	Reddish yellow; copious deposit of lithates.
27	1360	1015	"	"	16.400	1.800		" slight cloudy deposit.
28	600	1022	"	"	19.095	1.675		Yellowish-red colour; slight uric acid deposit.
29	670	1020	"	"	15.695	1.825		" cloudy.
30	780	1018	"	"				Reddish yellow; cloudy.



35	590	1017	"	14-045	1-325	3rd week of convales.	Yellowish red ; no deposit.
36	810	1015	"	15-795	2-025	"	" deposit of lithates.
37	800	1014	"	11-200	2-800	"	Reddish yellow ; cloudy ; no deposit.
38	Lost	1015	"	.....	.....	"	Yellowish red " "
39	610	1019	"	13-420	1-525	"	Reddish yellow " "
40	600	1015	"	12-300	1-800	"	" cloudy deposit.
41	Lost	1019	"	.....	.....	"	" lithates.
42	490	1018	"	10-780	2-205	"	Yellowish red ; copious deposit of lithates.
43	510	1017	"	9-180	3-570	"	Reddish yellow " "
44	910	1014	"	10-920	5-005	"	" no deposit.
45	880	1013	"	11-205	3-735	"	" "
46	1250	1015	"	15-000	6-875	"	Yellow ; cloudy.
47	1880	1011	"	15-040	10-340	"	" "
48	1310	1014	"	15-720	8-515	"	" "
49	1540	1013	"	16-940	8-470	"	" no deposit.
50	950	1017	"	14-250	5-225	"	" cloudy.
51	1680	1012	"	15-980	8-400	"	" "
52	1750	1012	"	16-625	8-750	"	" "
53	1350	1013	"	16-875	7-425	"	" "
54	1620	1011	"	16-380	7-290	"	" "
55	1440	1011	"	13-680	6-480	"	" "
56	1490	1011	"	14-900	8-195	"	Bright yellow ; cloudy deposit.
57	1510	1015	"	15-875	9-060	"	" "
58	1720	1011	"	18-065	10-320	"	" "
59	1050	1015	"	11-550	6-275	"	Pale " "
60	1630	1012	"	18-745	8-150	"	Bright " "

## CASE 2—continued.

Day of disease.	Quantity of water.	Specific gravity.	Reaction.	Albumen.	Urea.	Chlorides.	Colour and deposit.
	C. C.				Grammes.	Grammes.	
61	1390	1014	Acid	None	16·680	9·035	Bright yellow; cloudy deposit.
62	1100	1016	"	"	13·200	7·700	Pale yellow; slight deposit of phosphates on boiling.
63	1380	1013	"	"	15·180	8·970	Bright yellow; cloudy.
64	1210	1015	"	"	15·730	7·260	"
65	1150	1012	"	"	13·800	5·750	"
66	1590	1015	"	"	19·875	9·540	Bright yellow; cloudy.
67	920	1015	"	"	13·800	5·520	" slight deposit.
68	1350	1014	"	"	17·550	12·150	cloudy.
69	960	1018	"	"	16·320	8·640	"
70	1510	1015	"	"	18·120	13·590	"
71	1360	1013	"	"	16·320	10·880	"
72	1780	1019	"	"	24·030	19·580	Pale
73	1400	1018	"	"	18·900	.....	" slight cloudy deposit.
74	640	1023	"	"	12·800	6·400	"
75	1160	1018	"	"	18·560	11·020	" yellow lithates.
76	710	1026	"	"	17·750	7·455	Reddish yellow; lithates.
77	960	1024	"	"	.....	8·715	"
78	830	1020	"	"	19·120	10·545	Yellow; slight cloudy deposit.
79	1110	1020	"	"	22·200	.....	Reddish yellow; lithates.

In this case, therefore, the average daily excretion of urinary water, urea, and chlorides was—

		Amount of urine.	Urea.	Chlorides.
		C. C.	Grammes.	Grammes.
2	During the lysis .....	945	24·562	·895
	or per kilogramme of body weight .....	.....	·697	·0254
3	During the 1st week of convalescence ...	741	23·862	2·452
	or per kilogramme of body weight .....	.....	·677	·0696
4	During the 2nd week of convalescence ...	590	17·572	2·685
	or per kilogramme of body weight .....	.....	·499	·0762
5	During the 3rd week of convalescence ...	685	13·615	1·919
	or per kilogramme of body weight .....	.....	·386	·0545
6	During the 4th week of convalescence ...	765	11·564	3·865
	or per kilogramme of body weight .....	.....	·328	·111
7	During the 5th week of convalescence ...	1494	15·915	8·160
	or per kilogramme of body weight .....	.....	·451	·231
8	During the 6th week of convalescence ...	1494	15·599	7·953
	or per kilogramme of body weight .....	.....	·443	·225
9	During the 7th week of convalescence.....	1248	15·466	7·682
	or per kilogramme of body weight .....	.....	·439	·218
10	During the 8th week of convalescence ...	1285	17·720	11·873
	or per kilogramme of body weight .....	.....	·503	·337
11	During the 9th week of convalescence ...	95	19·407	9·433
	or per kilogramme of body weight .....	.....	·551	·268
	The normal excretion for a man æt. 51 would be per kilogramme of body weight.....	23	·450	·126

CASE 3.—Robert W—, æt. 22, admitted into London Fever Hospital October 7th, 1865, labourer; single. Has always had good health, and was working regularly up to the commencement of his present illness six days ago.

His attack commenced with headache, rigor, and general pains; it came on very suddenly, and he was obliged to give up work this first day of his illness. On admission the seventh day, his skin felt very hot and there was a copious,

rather dark, measly rash on the trunk and limbs. His tongue was covered with a thick clammy brownish fur. He was very restless, continually getting out of bed and wandering a good deal; very thirsty. He was dull and stupid, but could answer questions when roused.

*Rash.*—Copious, measly, and dusky, on admission: grew very dark and petechial as his disease advanced. It began to fade on the fifteenth day, but remains of it were more or less visible till the twenty-seventh day.

*Complexion.*—Dusky and flushed; the eyes much suffused and injected, and the lips and teeth covered with sordes till the fourteenth day. It then gradually improved, and was noted as clear and good on the twenty-second day. The expression, at first characteristically typhus, was noted as natural on the twenty-sixth day.

*Sleep.*—He slept very badly; wandered much and was very restless till fourteenth day. On the sixteenth he slept well. During early convalescence from sixteenth to twenty-fourth day he slept almost continually.

*Tongue.*—Covered with a thick clammy brown fur when first seen; was dry, brown, and hard, from the tenth to the fourteenth days, then gradually became moist and clean; quite clean on the twenty-first day.

*Bowels.*—Rather confined. On the ninth day had a simple soap-and-water enema, else opened naturally throughout attack.

*Headache, general pains, and thirst,* were not complained of after admission.

*Appetite.*—Noted as returning on the eighteenth day, was not good till the twentieth day.

*Mind.*—Much low, talkative, delirium, till the fourteenth day, then very drowsy, deaf, and stupid, this lasting more or less till the twenty-fourth day.

*Lungs.*—Slight dulness at bases from the twelfth to the fifteenth days. Sonorous râles audible over whole back, and at bases mucous râles from tenth to seventeenth days.



1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50	51	52	53	54	55	56	57	58	59	60	61	62	63	64	65	66	67	68	69	70	71	72	73	74	75	76	77	78	79	80	81	82	83	84	85	86	87	88	89	90	91	92	93	94	95	96	97	98	99	100	101	102	103	104	105	106	107	108	109	110	111	112	113	114	115	116	117	118	119	120	121	122	123	124	125	126	127	128	129	130	131	132	133	134	135	136	137	138	139	140	141	142	143	144	145	146	147	148	149	150	151	152	153	154	155	156	157	158	159	160	161	162	163	164	165	166	167	168	169	170	171	172	173	174	175	176	177	178	179	180	181	182	183	184	185	186	187	188	189	190	191	192	193	194	195	196	197	198	199	200	201	202	203	204	205	206	207	208	209	210	211	212	213	214	215	216	217	218	219	220	221	222	223	224	225	226	227	228	229	230	231	232	233	234	235	236	237	238	239	240	241	242	243	244	245	246	247	248	249	250	251	252	253	254	255	256	257	258	259	260	261	262	263	264	265	266	267	268	269	270	271	272	273	274	275	276	277	278	279	280	281	282	283	284	285	286	287	288	289	290	291	292	293	294	295	296	297	298	299	300	301	302	303	304	305	306	307	308	309	310	311	312	313	314	315	316	317	318	319	320	321	322	323	324	325	326	327	328	329	330	331	332	333	334	335	336	337	338	339	340	341	342	343	344	345	346	347	348	349	350	351	352	353	354	355	356	357	358	359	360	361	362	363	364	365	366	367	368	369	370	371	372	373	374	375	376	377	378	379	380	381	382	383	384	385	386	387	388	389	390	391	392	393	394	395	396	397	398	399	400	401	402	403	404	405	406	407	408	409	410	411	412	413	414	415	416	417	418	419	420	421	422	423	424	425	426	427	428	429	430	431	432	433	434	435	436	437	438	439	440	441	442	443	444	445	446	447	448	449	450	451	452	453	454	455	456	457	458	459	460	461	462	463	464	465	466	467	468	469	470	471	472	473	474	475	476	477	478	479	480	481	482	483	484	485	486	487	488	489	490	491	492	493	494	495	496	497	498	499	500	501	502	503	504	505	506	507	508	509	510	511	512	513	514	515	516	517	518	519	520	521	522	523	524	525	526	527	528	529	530	531	532	533	534	535	536	537	538	539	540	541	542	543	544	545	546	547	548	549	550	551	552	553	554	555	556	557	558	559	560	561	562	563	564	565	566	567	568	569	570	571	572	573	574	575	576	577	578	579	580	581	582	583	584	585	586	587	588	589	590	591	592	593	594	595	596	597	598	599	600	601	602	603	604	605	606	607	608	609	610	611	612	613	614	615	616	617	618	619	620	621	622	623	624	625	626	627	628	629	630	631	632	633	634	635	636	637	638	639	640	641	642	643	644	645	646	647	648	649	650	651	652	653	654	655	656	657	658	659	660	661	662	663	664	665	666	667	668	669	670	671	672	673	674	675	676	677	678	679	680	681	682	683	684	685	686	687	688	689	690	691	692	693	694	695	696	697	698	699	700	701	702	703	704	705	706	707	708	709	710	711	712	713	714	715	716	717	718	719	720	721	722	723	724	725	726	727	728	729	730	731	732	733	734	735	736	737	738	739	740	741	742	743	744	745	746	747	748	749	750	751	752	753	754	755	756	757	758	759	760	761	762	763	764	765	766	767	768	769	770	771	772	773	774	775	776	777	778	779	780	781	782	783	784	785	786	787	788	789	790	791	792	793	794	795	796	797	798	799	800	801	802	803	804	805	806	807	808	809	810	811	812	813	814	815	816	817	818	819	820	821	822	823	824	825	826	827	828	829	830	831	832	833	834	835	836	837	838	839	840	841	842	843	844	845	846	847	848	849	850	851	852	853	854	855	856	857	858	859	860	861	862	863	864	865	866	867	868	869	870	871	872	873	874	875	876	877	878	879	880	881	882	883	884	885	886	887	888	889	890	891	892	893	894	895	896	897	898	899	900	901	902	903	904	905	906	907	908	909	910	911	912	913	914	915	916	917	918	919	920	921	922	923	924	925	926	927	928	929	930	931	932	933	934	935	936	937	938	939	940	941	942	943	944	945	946	947	948	949	950	951	952	953	954	955	956	957	958	959	960	961	962	963	964	965	966	967	968	969	970	971	972	973	974	975	976	977	978	979	980	981	982	983	984	985	986	987	988	989	990	991	992	993	994	995	996	997	998	999	1000	1001	1002	1003	1004	1005	1006	1007	1008	1009	1010	1011	1012	1013	1014	1015	1016	1017	1018	1019	1020	1021	1022	1023	1024	1025	1026	1027	1028	1029	1030	1031	1032	1033	1034	1035	1036	1037	1038	1039	1040	1041	1042	1043	1044	1045	1046	1047	1048	1049	1050	1051	1052	1053	1054	1055	1056	1057	1058	1059	1060	1061	1062	1063	1064	1065	1066	1067	1068	1069	1070	1071	1072	1073	1074	1075	1076	1077	1078	1079	1080	1081	1082	1083	1084	1085	1086	1087	1088	1089	1090	1091	1092	1093	1094	1095	1096	1097	1098	1099	1100	1101	1102	1103	1104	1105	1106	1107	1108	1109	1110	1111	1112	1113	1114	1115	1116	1117	1118	1119	1120	1121	1122	1123	1124	1125	1126	1127	1128	1129	1130	1131	1132	1133	1134	1135	1136	1137	1138	1139	1140	1141	1142	1143	1144	1145	1146	1147	1148	1149	1150	1151	1152	1153	1154	1155	1156	1157	1158	1159	1160	1161	1162	1163	1164	1165	1166	1167	1168	1169	1170	1171	1172	1173	1174	1175	1176	1177	1178	1179	1180	1181	1182	1183	1184	1185	1186	1187	1188	1189	1190	1191	1192	1193	1194	1195	1196	1197	1198	1199	1200	1201	1202	1203	1204	1205	1206	1207	1208	1209	1210	1211	1212	1213	1214	1215	1216	1217	1218	1219	1220	1221	12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ounds very weak from the eighth till the nine-  
mpulse not to be felt during this time.  
s up for the first time on the twenty-eighth day,  
very weak indeed. He left the hospital on the  
y, feeling well and strong.  
d, on the twenty-ninth day, 44 kilogrammes or  
he thirty-ninth day, 57·3 kilogrammes or 111·5

*Treatment.*

℞ Æther mixture every four hours.  
Enema simplex.  
Mustard poultice to back.  
Quinine draught three times daily.  
Castor oil ʒss at once.

*Diet.*

Low diet with beef-tea.  
Brandy 6 ozs.  
Full diet and porter, 1 pint daily. To leave  
off brandy.  
Egg and extra bread. (See Diagram III.)



CASE 3.—Robert W—, æt. 22. *Analysis of Urine.*

Weight on 29th day . . 44 kilogrammes.

" 39th " . . 57.3 "

Day of disease.	Quantity of water.	Specific gravity.	Reaction.	Albumen.	Urea.	Chlorides.	Colour and deposit.			
							Grammes.	Before lysis.	During lysis.	1st week of convales.
10	G. C. 1760	1015	Acid	Trace	Grammes. 45.760	.....				
11	1800	1016	"	"	45.700	.....				
12	Lost	1016	"	"	.....	.....				
13	29.45	1007	"	"	51.637	.....				
14	3265	1005	"	"	42.445	.....				
15	1625	1015	"	"	32.825	.762				
16	1475	1015	"	"	44.250	.878				
17	685	1020	"	"	24.845	.842				
18	1125	1022	"	"	37.687	2.250				
19	985	1025	"	"	32.505	4.432				
20	820	1027	"	"	31.570	2.460				
21	760	1027	"	"	28.890	3.280				
22	655	1026	"	"	27.510	2.947				
23	620	1030	"	"	25.420	3.100				
24	630	1030	"	"	25.693	2.663				
25	700	1025	"	"	25.200	3.325				
26	Lost	1025	"	"	.....	.....				

Pale yellow, with deposit of lithates.

" "

" "

Bright yellow "

Reddish brown; copious deposit of lithates.

" "

Yellowish red, with slight cloudy deposit.

" no deposit.





In this case the average daily excretion of urinary water, urea, and chlorides has been—

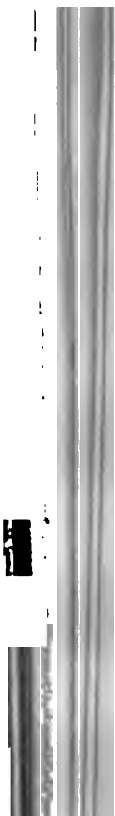
		Amount of urine.	Urea.	Chlorides.
		C. C.	Grammes.	Grammes.
1	Before the lysis .....	1760	45·760	Not ana-
	or per kilogramme of body weight .....	30·7	·799	lysed
2	During the lysis.....	2503	46·561	"
	or per kilogramme of body weight .....	45·2	·812	
3	During the following five days, during which the temperature was rather high.			
	1st week of convalescence .....	1159	34·322	1·632
	or per kilogramme of body weight .....	20·2	·599	·018
4	During 2nd week of convalescence .....	697	27·401	2·962
	or per kilogramme of body weight .....	12·1	·478	·051
5	During 3rd week of convalescence .....	1019	23·028	4·460
	or per kilogramme of body weight .....	17·6	·402	·078
6	During 4th week of convalescence .....	1579	26·105	12·323
	or per kilogramme of body weight .....	27·4	·455	·215
The normal amount for this man would be about .....		1501	33·180	8·21
or per kilogramme of body weight about .....		·23	·500	·126

CASE 4.—Samuel H—, æt. 38, admitted into the London Fever Hospital, October 10th, 1865, labourer by trade: married. He states that he has been out of work lately and not living well. His present illness commenced six days ago with headache, rigors, and general pains, and for the last two days he has been obliged to keep to his bed. With the exception of some castor oil, three days ago, he has not had any medicine.

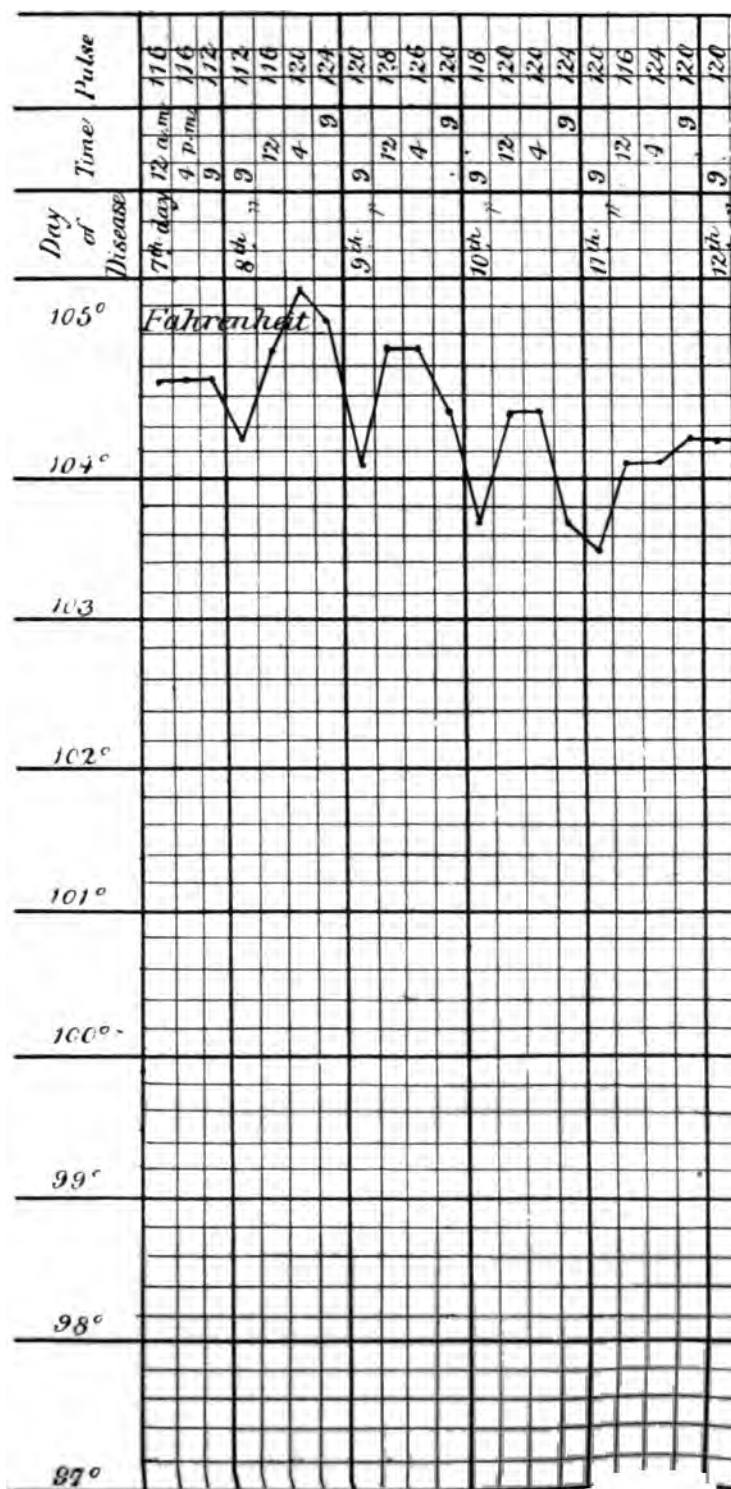
On admission his face was much flushed; his eyes were suffused and injected, the pupils small; his lips dry, with some sordes on the teeth; his expression dull and listless. He complained much of frontal headache and aching pain in his eyeballs. His skin was hot, with a distinct typhus rash. The tongue, moist at edges, was getting dry and hard down the centre; he was very thirsty. He had no cough, and his lungs were clear to the base.

*Rash*.—Distinct on admission, grew darker as the disease advanced. It began to fade on the fourteenth day, and was quite gone by the eighteenth day.









*Sleep.*—Restless and disturbed with more or less delirium, till the thirteenth day, afterwards good.

*Complexion.*—Dusky and flushed, with a heavy vacant expression till the thirteenth day, natural on the nineteenth day.

*Tongue.*—Was dry, red, and hard down the centre, till the eleventh day; on the twelfth day it was moist and covered with a yellowish creamy fur, and by the nineteenth day was quite clean.

*Bowels.*—Were rather confined throughout; patient had a simple enema on the thirteenth day, and took castor oil on the eighteenth day.

*Thirst.*—Very great till the sixteenth day; was not complained of afterwards.

*Appetite.*—Patient began to feel hungry on the seventeenth day; his appetite was very good on the nineteenth day.

*Headache.*—Intense at first, continued bad till the tenth day. None after the twelfth day.

*Lungs.*—Few; sonorous râles audible, chest and back from eleventh to sixteenth day. Patient did not complain of any cough.

*Heart.*—Sounds normal throughout; impulse not felt during second week's illness.

Patient was up for the first time on the twentieth day. He left the hospital feeling quite well on the twenty-eighth day.

He weighed, on the twentieth day, 50 kilogrammes or 110 lbs.; on the twenty-eighth 52 kilogrammes or 114·5 lbs.

#### *Treatment.*

7th day.	R	Æther mixture 3j every four hours.
13th	,,	Enema simplex.
18th	,,	Bark mixture 3j three times daily.
		Castor oil 3ij.

#### *Diet.*

7th day.	Low diet with beef-tea.
18th	,, Full diet and porter, 1 pint daily. (See Diag. IV.)

CASE 4.—Samuel H—, æt. 38. *Analysis of Urine.*  
 Weight on 20th day . . 50 kilogrammes.  
 " 28th day . . 52 "

Day of disease.	Quantity of water.	Specific gravity.	Reaction.	Albumen.	Urea.	Chlorides.	Colour and deposit.	
							Before lysis.	During lysis.
9	C. C. 955	1022	Acid	None	Grammes. 32.947	Grammes. .....		
10	1140	1020	"	"	35.340	.....		
11	850	1017	"	"	24.225	.....		
12	1150	1011	"	"	25.300	.575		
13	2440	1012	"	"	50.020	1.220		
14	820	1015	"	"	21.730	.615		
15	790	1016	"	"	22.120	.395		
16	2040	1015	"	"	49.980	2.040		
17	1900	1014	"	"	38.950	2.850		
18	1230	1014	"	"	27.675	2.460		
19	1045	1015	"	"	26.125	2.873		
20	640	1023	"	"	20.800	2.560		Reddish-brown colour, with copious deposit of lithates.
21	750	1018	"	"	22.875	3.750		Reddish yellow; some lithates.
22	470 <sup>1</sup>	1015	"	"	.....	.....		Pale yellow, with slight cloudy deposit.
23	1560	1012	"	"	24.960	6.240		Yellow colour. No deposit.
24	1760	1012	"	"	25.620	7.920		" "
25	1520	1015	"	"	22.800	8.260		" "
26	1560	1016	"	"	23.100	10.140		" "
27	990	1011	"	"	17.640	6.370		Deposit of lithates.
28	1680	1011	Alkaline	"	20.160	9.240		Bright yellow. No deposit.

<sup>1</sup> Some urine lost this day.



In this case the average daily excretion of urinary water, urea and chlorides, has been—

		Amount of water.	Urea.	Chlorides.
		C. C.	Grammes.	Grammes.
1	Before the lysis .....	1225	31·954	·808
	or per kilogramme of body weight .....	23·5	·605	·015
2	During the lysis.....	1401	32·970	2·123
	or per kilogramme of body weight .....	26·9	·634	·041
				(nearly)
3	During the 1st week of convalescence (8 days) .....	1306	22·282	6·810
	or per kilogramme of body weight .....	25·	·428	·131
The normal amount in this case would be per kilogramme of body weight about...		23	·500	·126

CASE 5.—W. B—, æt. 28, admitted into the London Fever Hospital, March 2nd, 1866. States that he has been ill five days.

On admission his skin was hot, and there was a distinct, rather dark typhus rash. His tongue was furred, and dry at the tip. The bowels had been open in the morning. He was complaining of much headache and pains all over him, and of great thirst. He had some cough, but his lungs were clear to bases. He was restless and wandering slightly.

*Rash.*—Copious on admission, grew dark and petechial as the disease advanced. It began to fade from the limbs on the eleventh day, from the trunk on the twelfth day, and was gone by the fifteenth day.

*Sleep.*—His nights were restless and disturbed, with much delirium till the twelfth day. On the thirteenth and afterwards he slept well.

*Aspect.*—Vacant and listless till twelfth day, good on the sixteenth day. Complexion muddy and sallow, with suffused and injected eyes, and slight sordes on lips and teeth till twelfth day.

*Tongue.*—Moist, and covered with a thick yellowish fur till the eleventh day. On the twelfth and thirteenth days it

was dry and brown, on the fourteenth improving, and on the sixteenth day was fairly clean.

*Bowels.*—Loose on seventh and eighth days, afterwards confined (by medicine) ; open freely by oil on eighteenth day.

*Headache.*—Very severe at first, was only occasional after the eighth day.

Nasty taste in mouth was much complained of till the thirteenth day.

*Thirst.*—Very great at first, was complained of slightly till the sixteenth day.

*Appetite.*—Noted a returning on the fourteenth ; was very good by the sixteenth day.

*Cough.*—Never very troublesome.

*Lungs.*—Few coarse crepitant râles, audible at left base from fourteenth to seventeenth days. No particular dulness at left base, local fremitus the same at both bases.

*Heart.*—Sounds normal, rather weak.

Patient got up for the first time on the twenty-second day. He left the Hospital feeling quite well and strong on the fortieth day.

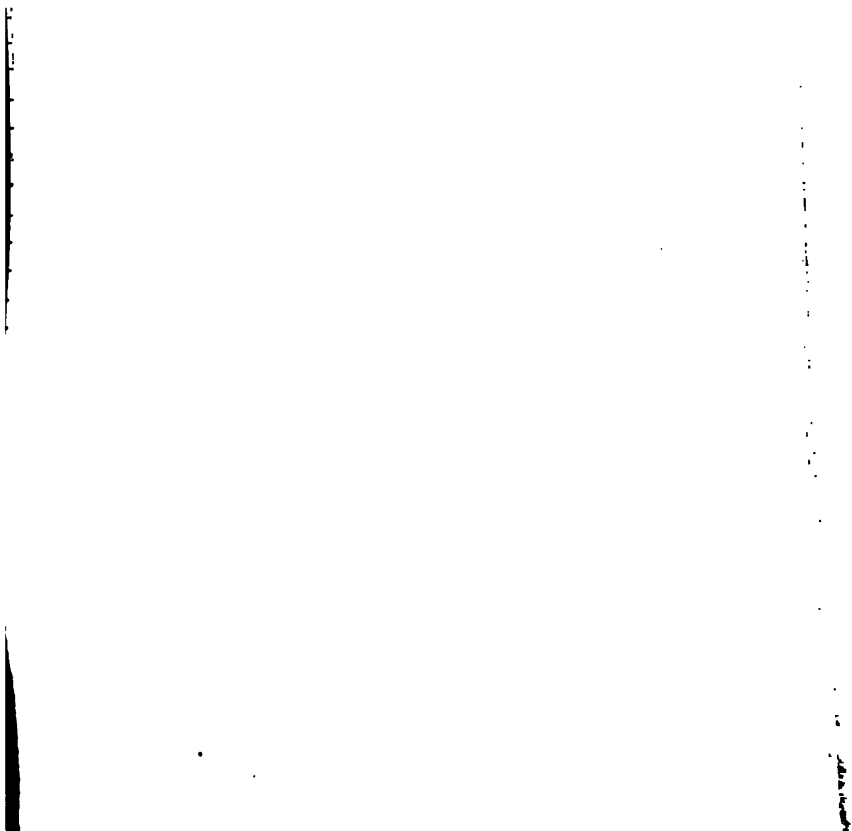
His weight on the thirty-fourth day was 52·05 kilogrammes, or 136·5 lbs. ; on the fortieth day was 63·4 kilogrammes, or 139·5 lbs.

#### *Treatment.*

6th day.	℞	Mist. Camphoræ ʒj, 4th horis.	Mustard poultice to back.
7th	„	Tinct. Catechu ʒj, Mist. Camph. ʒj,	three times daily.
11th	„	Mist. Camphoræ ʒj, 4th horis.	Repeat mustard.
14th	„	Bark mixture,	three times daily.
17th	„	Castor oil ʒj,	statim.

#### *Diet.*

6th day.	Low, beef-tea diet.
14th	„ Full diet. Porter Oj.
17th	„ One egg daily. (See Diagram V.)



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CASE 5.—William B—, æt. 28. *Analysis of Urine.*

Weight on 34th day . . 62 kilogrammes.  
 " 40th day . . 63·4 "

Day of disease.	Quantity of water.	Specific gravity.	Reaction.	Albumen.	Urea.	Chlorides.		Colour and deposit.
8	C. C. 590	1023	Acid	Slight trace	Grammes. 25·370	Grammes. ·147	Before lysis.	Yellowish red; copious lithates.
9	420 <sup>1</sup>	1027	"	"	17·220	·105	During lysis.	"
10	Lost	1024	"	None	Lost	Lost		"
11	1320	1018	"	"	48·840	·660	"	"
12	1320	1017	"	"	42·240	1·320		Reddish yellow;
13	940	1020	"	"	21·550	·940	"	"
14	560 <sup>1</sup>	1020	"	"	.....	.....	"	"
15	900	1022	"	"	29·700	1·350	"	Yellowish red; cloudy deposit; and some uric acid.
16	1070	1022	"	"	31·030	2·140	1st week of convalescence.	lithates.
17	960	1025	"	"	29·450	2·850		cloudy.
18	810 <sup>1</sup>	1025	"	"	.....	.....	"	deposit of lithates; and uric acid.
19	730 <sup>1</sup>	1020	"	"	.....	.....	"	"
20	960	1018	"	"	19·950	5·225	"	Reddish yellow; clear.
21	1560	1013	"	"	19·500	8·580	"	Yellow;
22	1500	1012	"	"	21·000	8·250	"	Reddish yellow, "

<sup>1</sup> Some urine lost on these days.

CASE 5—continued.

Day of disease.	Quantity of water.	Specific gravity.	Reaction.	Albumen.	Urea.	Chlorides.		Colour and deposit.
	C. C.				Grammes.	Grammes.		
23	1320	1017	Acid	None	21.945	9.310	2nd week of convalescence.	Reddish yellow; clear.
24	1060	1021	"	"	22.790	9.540		Yellow; "
25	1690	1017	"	"	28.560	12.600		Reddish yellow; slight deposit of lithates.
26	2320	1010	"	"	23.200	11.600		Bright yellow; clear.
27	1440	1017	"	"	27.360	10.080		Yellow; "
28	2160	1013	"	"	28.080	17.280		Bright yellow "
29	1300	1021	"	"	26.000	13.650		Yellow; "
30	1540	1017	"	"	27.720	15.400		"
31	2300	1016	"	"	30.800	18.800		Bright yellow; "
32	1560	1020	"	"	28.860	7.800		Yellow; clear; uric acid deposit.
33	1000	1022	"	"	26.000	8.500		" "
34	1540	1021	"	"	32.340	12.320		" "
35	2200	1013	"	"	27.500	15.510		Bright yellow; clear; uric acid deposit.
36	2170	1016	"	"	.....	19.530		" "
37	2680	1010	"	"	.....	16.080		" "
38	2020	1016	"	"	.....	14.140		Yellow; "
39	1430	1019	"	"	.....	12.870		" "
40	2350	1012	"	"	.....	16.450	4th week of conv.	" "

The average daily excretion of urinary water, urea, and chlorides during each period has been—

		Amount of water.	Urea.	Chlorides.
		C. C.	Grammes.	Grammes.
1	Before lysis (only one day's urine ..... or per kilogramme of body weight .....	590 .....	25·370 ·400	·147 .....
2	During lysis (four days' urine)..... or per kilogramme of body weight .....	1120 .....	35·582 ·561	·875 ·001
3	During 1st week of convalescence ..... or per kilogramme of body weight .....	1206 .....	24·186 ·381	5·409 ·085
4	During 2nd week of convalescence ..... or per kilogramme of body weight .....	1611 .....	24·410 ·400	12·008 ·189
5	During 3rd week of convalescence ..... or per kilogramme of body weight .....	1758 .....	28·870 ·453	13·980 ·220
6	During 4th week of convalescence ..... or per kilogramme of body weight .....	2120 .....		14·885 ·234
The normal rate per kilogramme is about.		1500	·500	·126

**CASE 6.**—William B—, æt. 27, admitted into the London Fever Hospital, November 7th, 1865. Is a strong healthy-looking man, and has never been laid up with any serious illness. His present attack commenced six days before his admission with headache, rigors, and general pains. When first seen, November 7th (seventh day of his illness), his skin was hot, and there was a copious rather dark typhus rash on trunk and limbs. His tongue was moist and covered with a thick creamy fur. He was restless and wandering slightly. His face was dusky and flushed. He did not complain of any pain, but was very thirsty.

**Rash.**—Copious and dark on admission, became quite petechial in character towards the end of the disease. It began to fade the twelfth day, and was quite gone by the fifteenth day.

**Sleep.**—Restless and disturbed, with much delirium till the twelfth day. Slept well on thirteenth.

**Complexion.**—Dusky and flushed with the suffused and injected eyes, and dry lips slightly covered with sordes till

the eleventh day: clearer on the twelfth and natural on the sixteenth day. The expression at first vacant and listless, was good on the sixteenth day.

*Tongue*.—Dry and brown on the ninth and tenth days, moist and thickly furred on the eleventh day. Was quite clean by the seventeenth day.

*Bowels*.—Open daily, rather relaxed at first.

*Headache*.—Not complained of after admission.

*Thirst*.—Great to the thirteenth day.

*Appetite*.—Returning on fourteenth day, good on sixteenth day.

*Mind*.—Confused and stupid, with some deafness to the tenth day. Pretty clear on thirteenth day.

*Lungs*.—Few bronchitic râles audible at bases tenth and eleventh days; no dulness on percussion elsewhere. Respiration was normal.

*Heart*.—Sounds weak but normal throughout. Impulse could not be felt during second week.

*Perspiration*.—More or less free from the fifteenth to the thirtieth days, with some sudaminæ on the chest and abdomen.

During convalescence patient suffered from boils and occasional sore throat, which caused sudden and high elevations of temperature.

Patient was up for the first time on the thirtieth day. He left the Hospital the thirty-sixth day, feeling well and strong.

His weight on the thirtieth day was 54·5 kilogrammes, or 120 lbs.; on the thirty-sixth day was 58 kilogrammes, or 127·5 lbs.

#### *Treatment.*

- |          |   |  |
|----------|---|--|
| 7th day. | R | Mist. Camphoræ 3j, 4th horis. Sedative draught at night.                       |
| 12th     | „ | Bark mixture, three times daily.   |
| 20th     | „ | Poultice to boil on buttock.   |
| 22nd     | „ | Quinine draught, three times daily. Gargarisma aluminis to be frequently used. |





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*Diet.*

- r. Low beef-tea diet.  
Full diet, and porter Oj.  
Low beef-tea diet and 3 oz. of wine.  
Fish and middle diet. To leave off wine.  
Chop and middle diet.  
Low beef-tea diet.  
Fish and middle diet. (See Diagram VI.)

CASE 6.—William B—, æt. 27. *Analysis of Urine.*

Day of disease.	Quantity of water.	Specific gravity.	Reaction.	Albumen.	Urea.	Chlorides.		Colour and deposit.
	C. C.				Grammes.	Grammes.		
9	Lost	1015	Acid	Slight trace	.....	.....	Before	Reddish yellow; cloudy deposit.
10	1385	1017	"	None	23.887	.682	lys.	Yellowish red; cloudy.
11	Lost	1011	"	"	.....	.....		Reddish yellow; cloudy with deposit.
12	1130	1017	"	"	37.290	.567		" no deposit.
13	970	1021	Neutral	"	34.435	.485		" lithates.
14	1050	1018	Acid	"	32.550	.525		Yellow; no deposit.
15	1060	1018	"	"	26.500	1.325	During	Reddish yellow; no deposit.
16	820	1018	"	"	22.140	1.435		Yellow; cloudy deposit.
17	1070	1017	"	"	33.705	2.675		"
18	1270	1017	"	"	33.020	5.715		Yellowish red; slight cloudy deposit.
19	830	1023	"	"	26.660	6.225		"
20	730	1022	"	"	23.360	2.920	1st week of	Reddish yellow; cloudy deposit; phosphates.
21	860	1023	"	"	27.950	5.160	convalescence.	" no deposit; phosphates less.
22	1140	1019	"	"	26.220	6.840		"

23	Limited	1012	"	"	.....	6°000	End week of convalescence.	"	cloudy deposit.
24	1200	1016	"	"	23°800	8°000		"	"
25	890	1018	"	"	15°130	8°900		Yellow; cloudy deposit, with few phosphates.	
26	1040	1023	"	"	30°180	5°200		Reddish yellow; deposit of lithates.	
27	1380	1016	"	"	29°260	6°660		"	cloudy deposit.
28	1330	1015	"	"	22°510	3°325		Yellow; no deposit.	
29	1270	1015	"	"	23°695	8°890		"	slight cloudy deposit.
30	1560	1012	"	"	18°720	9°360		Pale yellow; slight cloudy deposit.	
31	1570	1015	"	"	20°410	12°560		Bright "	"
32	1550	1011	"	"	19°375	7°750		Yellow; no deposit.	
33	1400	1011	"	"	21°600	°700		"	"
34	1230 <sup>1</sup>	1017	"	"	13°530 <sup>1</sup>	1°845		Bright yellow; slight deposit.	
35	2020	1010	"	"	21°210	8°080		"	"
36	1950	1011	"	"	23°400	13°650		"	"

<sup>1</sup> Some urine lost this day.

The average daily excretion of urinary water, urea, and chlorides during each period has been—

	Amount of water.	Urea.	Chlorides.
	C. C.	Grammes.	Grammes.
1 Before lysis (only 1 day's urine obtained)....	1365	23·887	·682
or per kilogramme of body weight .....	.....	·412	·0117
2 During lysis (4 days' urine).....	1052	32·694	·723
or per kilogramme of body weight .....	.....	·563	·012
3 During the 1st week of convalescence .....	960	27·565	4·424
or per kilogramme of body weight .....	.....	·475	·076
4 During the 2nd week of convalescence .....	1009	24·442	6·486
or per kilogramme of body weight .....	.....	·440	·112
5 During 3rd week of convalescence .....	1841	20·786	10·408
or per kilogramme of body weight .....	.....	·358	·179

CASE 7.—Louis S—, æt. 21, admitted into the London Fever Hospital January 1st, 1866; is a labourer. Has always enjoyed very good health; he has been ill eight days; says that his attack commenced with rigor, headache, and general pains, felt at first as if he had a very bad cold.

On admission (ninth day of his illness) his skin was very hot, and there was a copious, dark, typhus rash on trunk and limbs; his tongue was dry and brown down the centre; he was very thirsty; he did not complain of any pain, and he was rather deaf and stupid.

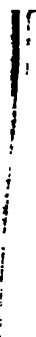
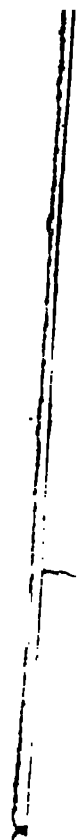
*Rash.*—Copious and dark on admission, became quite petechial about the end of the second week. It began to fade on the eleventh day, but was not entirely gone till the nineteenth day.

*Sleep.*—Restless and disturbed with occasional moaning and some delirium till the twelfth day, better afterwards.

*Complexion and Expression.*—Characteristic of typhus, with much sordes on the lips and teeth. Began to improve on the fifteenth day, and was natural on the twentieth day.

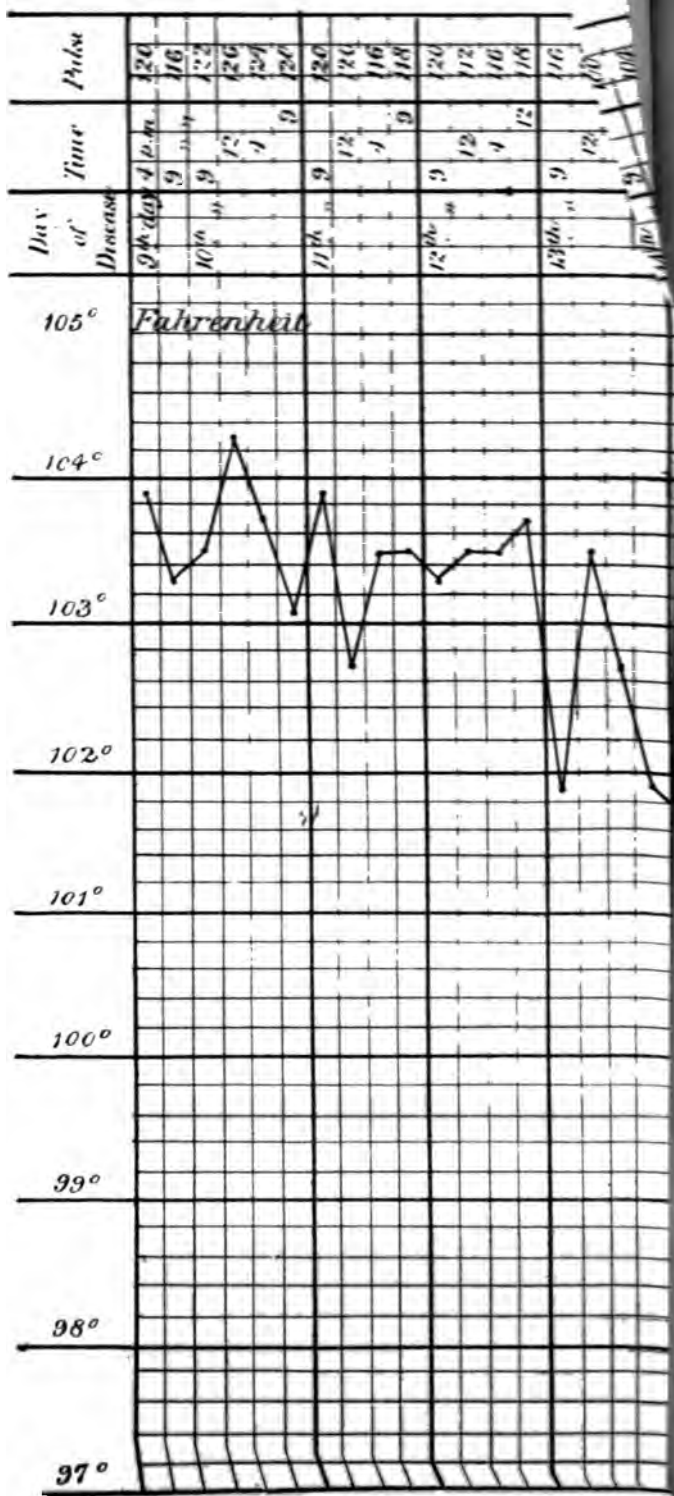
*Tongue.*—Dry and brown down the centre till the eleventh day; covered with a thick yellowish clammy fur on the twelfth day, cleaning on the thirteenth day, clean on the nineteenth day.











*Bowels.*—Rather loose at first, were open daily during acute stage.

*Headache.*—None after admission.

*Thirst.*—Very great at first, was complained of more or less till the nineteenth day.

Nasty taste in mouth, very bad till the twelfth day.

*Appetite.*—Returning on the seventeenth day, good on the nineteenth day.

*Mind.*—Very dull and stupid, and very deaf till the fourteenth day; he continued heavy and restless, sleeping almost all day and night, with exception of meal times, till seventeenth day, then was much more sensible and scarcely at all deaf.

*Cough.*—Very troublesome between the twelfth and fifteenth days.

*Lungs.*—Numerous sonorous and large mucus râles were audible at bases from the tenth to the seventeenth day, and more or less sonorous râles over whole back, slight dulness on percussion at bases.

*Heart.*—Sounds very weak, and impulse indistinct till the seventeenth day.

Patient was up for the first time on the twenty-sixth day. He was discharged the thirty-third day feeling quite well and strong.

His weight on the nineteenth day was 47·5 kilogrammes or 104·5 lbs; on the thirty-third day was 56 kilogrammes or 123 lbs.

#### *Treatment.*

9th day.	R	Æther draught 3j, 4tis horis.
12th	„	Mustard to back.
15th	„	Repeat mustard poultice.
19th	„	Bark mixture three times daily.
21st	„	Castor oil 3j, at once.

#### *Diet.*

9th day.	Low diet, with beef-tea, brandy 4 oz.
19th	„ Fish and middle diet; porter Oj.
27th	„ Full diet. (See Diagram VIII.)

CASE 7.—Lewis S—, æt. 21. *Analysis of Urine.*

Day of disease.	Quantity of water.	Specific gravity.	Reaction.	Albumen.	Urea.	Chlorides.		Colour and deposit.
	C. C.				Grammes.	Grammes.	Before lysis.	
11	Lost	1021	Acid	Trace	.....	.....		Reddish yellow; clear.
12	1200	1018	"	"	39.600	Trace		" " no deposit.
13	1240	1016	"	"	37.820	"		" " "
14	1510	1019	"	None	49.830	"		Yellowish red, with lithæta.
15	1050	1022	"	"	36.750	"		" " "
16	1480	1020	"	"	54.020	"		" clear; no deposit.
17	980	1024	"	"	34.300	.980		" " "
18	1540	1013	"	"	36.190	5.890		" " "
19	1940	1013	"	"	37.860	5.820		Reddish yellow; clear.
20	1500	1010	"	"	30.000	4.500		Yellow " "
21	1190	1017	"	"	26.180	5.950		" " "
22	930	1018	"	"	26.970	3.720		Yellowish red " light cloudy deposit.
23	780	1019	"	"	23.400	3.120		" " clear.
24	1340	1017	"	"	32.820	6.080		" " "
25	1460	1017	"	"	33.580	8.080		Yellow; slight cloudy deposit.
26	1790	1013	"	"	32.220	8.950	Last week of convalescence.	

Patient's weight on the 9th day was 47·5 kilogrammes or 104·5 lbs.; on the 33rd day was 56 kilogrammes or 123 lbs.

The average daily excretion of urinary water, urea, and chlorides during each period has been—

	Amount of water.	Urea.	Chlorides.
	C. C.	Grammes.	Grammes.
1 Before lysis (2 days' urine) .....	1220	38·710	Trace
or per kilogramme of body weight .....	.....	·691	
2 During lysis .....	1417	41·491	4·063
or per kilogramme of body weight .....	.....	·741	·0725
3 During the 1st week of convalescence.....	1284	29·310	5·757
or per kilogramme of body weight .....	.....	·523	·1028
The normal excretion per kilogramme of body weight would be about .....	.....	·005	·126

**CASE 8.**—George W—, æt. 18, labourer, admitted into the London Fever Hospital October 25th, suffering from scarlet fever; from this attack he began to coalesce October 30th, and was removed to the Convalescent Scarlet Ward on November 3rd. Whilst there, after he had quite recovered from his attack of scarlet fever, he contracted typhus fever. Says that he did not feel so well on November 25th, he felt cold and chilly, and had some headache and general aching pains; he gradually became worse, his appetite entirely failed, he complained of great thirst and passed bad and restless nights.

On the 29th, fifth day of disease, a distinct typhus rash was visible on the backs of hands and arms, and slightly on the trunk; his headache and general pains, though better, were still bad, he complained of much thirst; his tongue was moist with white fur on dorsum, red at tips and edges; the bowels had been opened daily, not at all loose; the lungs and heart sounds were normal, and his mind quite clear.

**Rash.**—On the fifth day the rash was copious on the backs of hands and arms, slight on the trunk. On the sixth day a

copious rash was everywhere visible, and it gradually grew darker as the disease advanced. It began to fade on the ninth day and was entirely gone on the fifteenth day.

*Sleep.*—He passed restless and disturbed nights up to the 16th, with little low muttering delirium each night; on the 15th he was disturbed by noisy patients in the same ward and wandered much; on the 17th and afterwards he slept well.

*Complexion.*—Was muddy and flushed, with suffused and injected eyes and more or less sordes on lips and teeth till thirteenth day; then gradually improved, and on the 20th it was noted as quite clear. Expression characteristically typhus at first, gradually cleared up, and was natural on the twentieth day.

*Tongue.*—Moist and red at tips and edges with white fur on dorsum, at first, was dry, and brown on the eighth day and continued so to the sixteenth; was quite clean on the nineteenth day.

*Bowels.*—Were open throughout without medicine, rather loose on the eighth day.

*Headache and general pains* intense at first, were not complained of after the sixth day. He complained much of nasty taste in his mouth till the eighth day.

*Mind.*—Patient was in a low heavy and stupid state from the ninth to the fifteenth day; on the nineteenth his mind was pretty clear, and he could answer questions readily.

*Appetite.*—Noted as returning on the seventeenth, and was very good on the nineteenth day.

*Cough.*—Slight from the thirteenth to the twenty-first day.

*Lungs.*—Few bronchitic râles with some mucous râles at bases audible at the end of the second and beginning of the third week. Quite clear on the twenty-second day.

*Heart.*—Sounds normal throughout. The impulse and first sound rather weak from the eighth to the fifteenth days.

Patient got up for the first time on the twenty-sixth day and convalesced rapidly afterwards.







patient's weight on the twenty-seventh day, was 93 lbs. or kilogrammes; on the thirty-seventh day, 102·5 lbs. or kilogrammes; on the fiftieth day, 113 lbs. or 51·4 kilogrammes.

*Treatment.*

h day.	℞	Mist. Camphoræ ʒj, 4tis horis.
h „		Mist. Cretæ cum Catechu ʒj subinde.
h „		Mustard to back.
h „		Haust. Cinchonæ ʒj, ter die.

*Diet.*

h day.	Low, beef-tea.
h „	Full diet and porter Oj.

(See Diagram VIII.)

CASE 8.—George W—, æt. 18. *Analysis of Urine.*

Day of disease.	Quantity of water.	Specific gravity.	Reaction.	Albumen.	Urea.	Chlorides.		Colour and deposit.
	C. C.				Grammes.	Grammes.		
7	740	1028	Acid	None	26.640	2.930	Before lysis.	Yellowish red, with slight cloudy deposit.
8	470 <sup>1</sup>	1029	"	Trace	16.920	.940		"
9	470 <sup>1</sup>	1023	"	None	16.920	.235		" no deposit.
10	1110	1021	"	"	34.410	.555		" cloudy deposit.
11	860	1019	"	"	24.940	Trace		Reddish yellow; no deposit.
12	1115	1020	"	"	35.680	"		"
13	1300	1016	"	"	31.220	.650		Yellow; cloudy; no deposit.
14	1570	1012	"	"	25.120	3.925	During lysis.	"
15	870 <sup>1</sup>	1018	"	"	20.010	1.305		"
16	910	1025	"	"	25.935	1.365		Reddish yellow; deposit of lithates.
17	Lowt	1023	"	"	.....	.....		" cloudy; no deposit.
18	470	1027	"	"	16.110	1.645		" deposit of lithates.
19	960	1018	"	"	19.200	5.280		Yellow; slight cloudy deposit.
20	780 <sup>1</sup>	1016	"	"	14.820	1.950		" cloudy, with deposit.
21	740	1020	"	"	16.650	2.960		" deposit of lithates; and some phosphates.
22	650	1017	"	"	13.650	2.600		Reddish yellow; no deposit.
23	730	1016	"	"	13.870	2.920	1st week of convalescence.	"

24	590	1022	"	"	14:160	8:245	2nd week of convalescence.	"	copious deposit of lithates.
25	640	1020	"	"	13:760	3:200	"	"	no deposit.
26	400	1028	"	"	13:200	2:800	"	"	"
27	500		"	"					
28	650	1024	"	"	16:250	5:850		Yellowish red, with cloudy deposit.	
29	680	1023	"	"	12:760	5:220		Reddish yellow; deposit of lithates.	
30	920	1017	"	"	15:640	5:080		Yellow; cloudy deposit.	
31	820	1015	"	"	18:940	4:100		"	"
32	710	1020	"	"	13:845	4:615		"	no deposit.
33	660	1015	"	"	12:880	3:960		"	"
34	830	1015	"	"	14:110	5:810		"	"
35	1190	1011	"	"	14:280	5:950		Pale yellow; cloudy; no deposit.	
36	1170	1012	"	"	14:625	4:680		"	"
37	1170	1011	"	"	14:625	4:095		Bright yellow; no deposit.	
38	1200	1010	"	"	12:600	6:000		"	light deposit.
39	1760	1010	"	"	21:120	7:900		"	no deposit.
40	1310	1008	"	"	15:720	6:550		"	"
41	2150	1010	"	"	19:350	8:600		"	"
42	1400	1016	"	"	23:100	10:500		Yellow: clear.	
43	2150	1010	"	"	24:725	9:675		"	"
44	2140	1015	"	"	24:610	19:260		Bright yellow; clear.	
45	1990	1012	"	"	22:885	17:910		"	"
46	2530	1013	"	"	27:830	25:300		"	"
47	1700	1012	"	"	27:200	14:450		"	"
48	1570	1015	"	"	22:765	10:205		Yellow	"
49	1330	1016	"	"	23:940	9:975		"	"

<sup>1</sup> Some of urine lost each of these days so marked.

Weight on the 27th day 43·3 kilogrammes or 93 lbs.; on the 50th day 51·4 kilogrammes or 113 lbs.

The average daily excretion of urinary water, urea, and chlorides during each period has been—

		Amount of water.	Urea.	Chlorides.
		C. C.	Grammes.	Grammes.
1	Before lysis (4 days' urine) .....	·956	31·667	1·757
	or per kilogramme of body weight .....	.....	·616	·0339
2	During lysis .....	·1260	25·569	1·811
	or per kilogramme of body weight .....	.....	·497	·0352
3	During 1st week of convalescence .....	·710	15·717	2·892
	or per kilogramme of body weight .....	.....	·305	·0523
4	During 2nd week of convalescence .....	·611	14·295	4·202
	or per kilogramme of body weight .....	.....	·278	·0822
5	During 3rd week of convalescence .....	·935	14·043	4·744
	or per kilogramme of body weight .....	.....	·282	·0923
6	During 4th week of convalescence .....	·1730	20·175	9·783
	or per kilogramme of body weight .....	.....	·392	·190
7	During 5th week of convalescence .....	·1824	24·924	15·568
	or per kilogramme of body weight .....	.....	·485	·302
The average normal excretion per kilo- gramme of body weight would be about		.....	·500	·126

CASE 9.—Thomas S—, æt. 13. Errand boy. Admitted into the London Fever Hospital on March 14th. States that he has been ill for two days. The attack commenced whilst at work with headache, rigors, and general pains. He has always enjoyed good health, and is a well-made healthy looking boy.

On admission patient was still suffering much from headache, and pains in back and limbs. His mind was quite clear. His skin was hot and mottled, but there was no distinct typhus rash. The tongue was moist and thickly furred. The bowels were open the day before admission. The lungs and heart were quite healthy. He was rather restless, face was slightly flushed, and he complained of much thirst.

*Rash.*—A copious typhus rash appeared on the fifth day, which grew dark and petechial as the disease advanced. It began to fade on the tenth, and was quite gone on the fifteenth day.

*Sleep.*—He slept badly, and was very restless, with more or less delirium till the ninth day; on the tenth day he slept better, but on the eleventh, being disturbed by a wildly delirious patient in the same ward, the delirium returned, and continued with more or less severity till the thirteenth day.

*Complexion.*—Dusky and flushed, with suffused and injected eyes, and some slight sordes on lips and teeth till the thirteenth day. Quite clear on the eighteenth day.

*Expression.*—Dull, vacant, and listless at first, began to improve on the thirteenth, and was quite natural on the eighteenth.

*Tongue.*—With the exception of the twelfth day, when it was dry and brown down the centre, the tongue was moist throughout, thickly coated with yellowish creamy fur during the early part of the disease. First noted as “clean” on the seventeenth day.

*Bowels.*—Open, without medicine, throughout; were rather loose from the fifth to the seventh days, and on the eleventh and twelfth days.

*Headache.*—Intense at first, lasted more or less till the seventh day. None afterwards.

Nasty taste in mouth much complained of till the ninth day.

*Appetite.*—Noted as returning on the fifteenth, good on the nineteenth day.

*Cough.*—Rather troublesome from the fifth to the seventeenth days. Expectoration frothy, and slightly mucopurulent.

*Lungs.*—Never any dulness chest or back. Numerous sonorous and sybilant râles audible, chiefly at back, from fifth to seventeenth days.

*Heart.*—Sounds were healthy throughout; rather weak at first.

Patient got up for the first time on the twentieth day.

*Weight.*—On the twenty-sixth day patient weighed 32·5 kilogrammes, or 71·5 lbs.; on the thirtieth day he weighed 34·5 kilogrammes, or 76 lbs.

*Treatment.*

3rd day.	℞	Mist. Camphoræ ʒj, every four hours.
5th	„	Mist. Cretæ ʒj, Tinct. Catechu ʒj. Three times daily.
8th	„	Mustard poultice to back. Mist. Camphoræ as before.
11th	„	Rep. Haust. Cretæ and Catechu.
13th	„	Rep. Mustard poultice to back and chest.
15th	„	Hydrochlorate of Cinchonine gr. 1, Dilute Hydrochloric acid ℥x, Treacle ʒss, Aqua ad ʒj. Fiat haust., t. d. s.

*Diet.*

3rd day.	Low, beef-tea.
15th	„ Middle diet and fish.
20th	„ Full diet, and porter Oj. (See Diagram IX.)









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CASE 9.—Thomas S—, *et. 13. Analysis of Urine.*

Day of disease.	Quantity of water.	Specific gravity.	Reaction.	Albumen.	Urea.	Chlorides.		Colour and deposit.
	C. C.				Grammes.	Grammes.		
5	630	1031	Acid	None	26.460	1.260	Before lysis.	Reddish yellow, with lithates.
6	490	1029	"	"	20.580	.735		" "
7	790	1025	"	"	27.650	.790		" "
8	1020	1019	"	"	28.560	.610		Yellowish red; clear; no deposit.
9	1100	1015	"	"	28.050	.550		" "
10	Lost	1014	"	"	.....	.....		" "
11	830	1015	"	"	22.410	.415		Reddish yellow; clear.
12	670	1016	Alkaline	"	17.420	.335	During lysis.	" copious deposit of lithates.
13	Lost	1016	"	"	.....	.....		" "
14	"	1010	Acid	"	.....	.....		Yellow; clear; no deposit.
15	800	1013	"	"	13.600	3.200		" cloudy.
16	1050	1010	"	"	15.225	1.050		" clear.
17	930	1016	"	"	20.460	3.720		" cloudy.
18	1160	1015	Alkaline	"	20.880	5.800		Bright yellow; cloudy.
19	1080	1016	Acid	"	21.800	5.450	1st week of convalescence.	" "
20	1100	1016	Alkaline	"	23.100	4.400		" "
21	1370	1011	"	"	21.235	4.110		Yellow; clear.
22	1340	1011	Neutral	"	.....	5.360		" slightly cloudy.
23	1740	1009	Acid	"	.....	4.350		Bright yellow; clear.
24	1320	1007	"	"	.....	3.960		" "
25	1560	1010	"	"	.....	6.240	2nd week of conval.	cloudy.
26	1740	1008	"	"	19.140	6.960		Pale yellow
27	1660	1009	"	"	18.600	4.650		" "

Weight on the 26th day 32·5 kilogrammes or 71·5 lbs. ; on the 30th day 34·5 kilogrammes or 76 lbs.

The average daily excretion of urinary water, urea, and chlorides has been in each period—

		Amount of water.	Urea.	Chlorides
		C. C.	G. mmes.	Grammes.
1	Before lysis '4 days' urine) .....	·732	25·812	·823
	or per kilogramme of body weight .....	.....	·748	·0239
2	During lysis .....	·934	19·712	2·153
	or per kilogramme of body weight .....	.....	·571	·0624
3	During 1st week of convalescence .....	·1326	22·045	4·771
	or per kilogramme of body weight .....	.....	·639	·138
4	During 2nd week of convalescence .....	·1720	18·870	5·283
	or per kilogramme of body weight .....	.....	·547	·153

CASE 10.—Joseph L—, æt. 17, admitted into the London Fever Hospital, October 30th, 1865. A strong well-made boy. Works at brick-laying. His present illness commenced seven days before admission with pain in his chest, and shortness of breath, followed by shivering, headache, and pains all over.

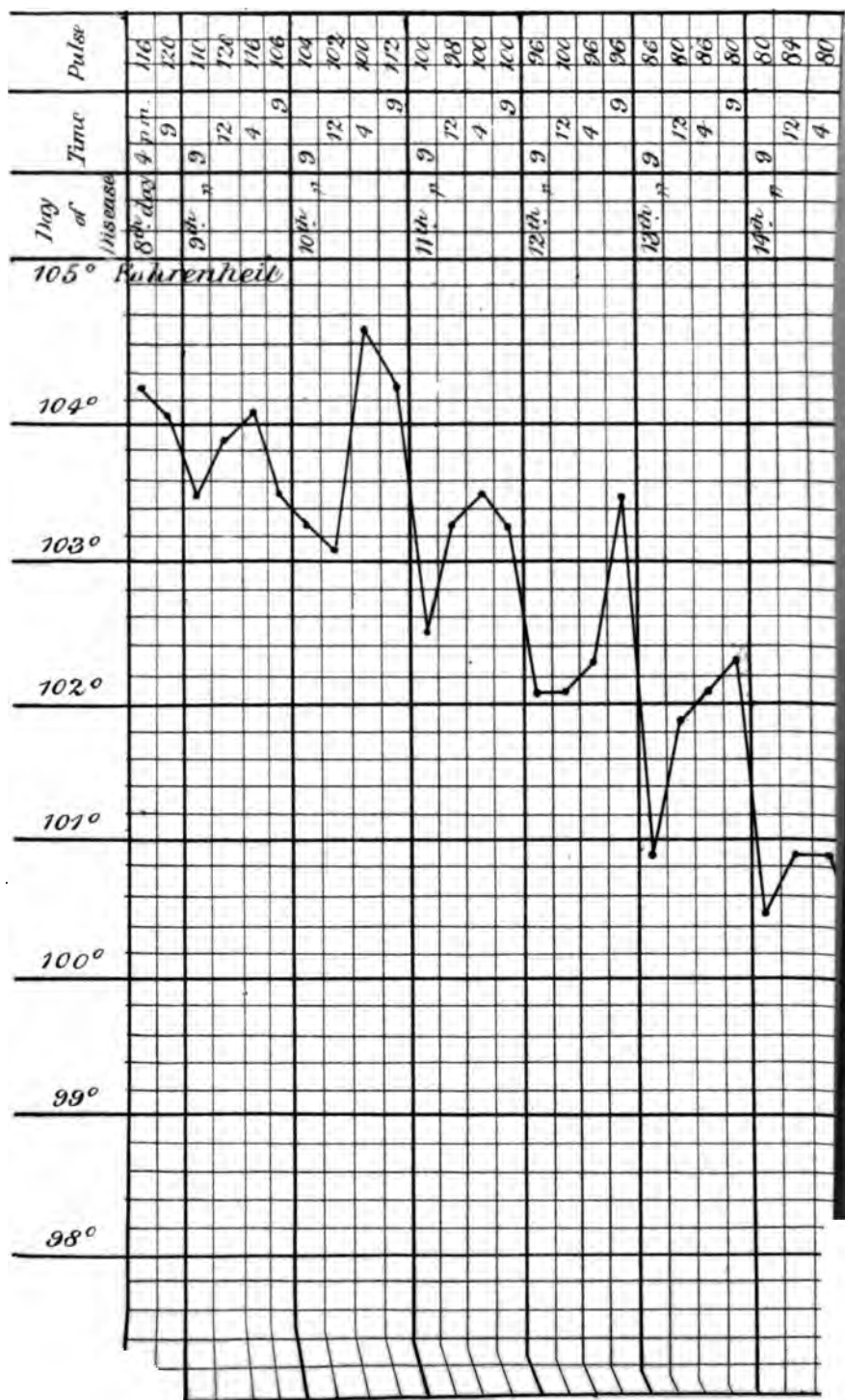
On admission his skin was hot, and covered with a pinkish typhus rash. His face was much flushed, and his expression rather dull. His tongue was moist and furred down the centre. His bowels had not been open for two days. He was complaining much of headache and thirst. He had a slight cough, but the lungs were clear to bases, and the heart sounds were normal.

*Rash.*—Gradually became darker; it began to fade on the twelfth, and was quite gone by the seventeenth day.

*Sleep.*—Restless and disturbed, with much delirium, from the tenth to the thirteenth days; he slept well on the fourteenth day, and afterwards.

*Complexion.*—Dusky and flushed at first; was pallid on the tenth, and not clear and good till the twentieth. No sordes on lips throughout. Eyes much suffused and injected





at first. The expression, dull to the thirteenth, was good by the eighteenth day.

*Tongue*.—Was dry and brown from the ninth to the fourteenth days. It was moist at edges on the fifteenth, and by the eighteenth was quite clear.

*Bowels*.—Confined generally; were opened by castor oil on the eleventh day. On the twenty-first day they were rather loose.

*Thirst*.—Very great, and much complained of till the twelfth day.

*Appetite*.—Noted as returning on the fourteenth, was good on the sixteenth day.

*Headache*.—Very bad indeed till the eleventh day, afterwards not complained of.

*Mind*.—Much confused till the fourteenth day. He was very deaf, and could not, when roused, answer questions truly up to the fourteenth day. Deafness lasted more or less till the eighteenth day.

*Cough*.—Slight throughout; a few bronchitic râles were audible at bases from the eighth to the thirteenth day.

*Heart*.—Sounds normal throughout.

Patient was up for the first time on the twentieth day. He left the Hospital feeling well and strong on the thirty-fifth day.

He weighed on the twenty-second day 35·7 kilogrammes, or 78·5 lbs.; on the thirty-eighth day 40 kilogrammes, or 88 lbs.

#### *Treatment.*

8th day.	℞	Mist. Camphoræ ʒj, every four hours.
11th	„	Castor oil ʒj, at once. Mustard to back.
15th	„	Bark mixture ʒj, three times daily.
21st	„	Chalk and Catechu mixture every now and then.

#### *Diet.*

8th day.	Low diet, with beef-tea.
15th	„ Full diet and porter Oj.
21st	„ One egg. (See Diagram X.)

CASE 10.—Joseph L—, æt. 17. *Analysis of Urine.*

Day of disease.	Quantity of water.	Specific gravity.	Reaction.	Albumen.	Urea.	Chlorides.		Colour and deposit.
	C. C.				Grammes.	Grammes.		
10	910	1018	Acid	None	26.390	1.365	Before	Yellowish red; clear.
11	1140	1014	"	"	26.225	1.710	lysis.	Reddish yellow; no deposit.
12	925	1014	"	"	19.887	1.166		"
13	790	1016	"	"	15.010	.790		"
14	1365	1008	"	"	17.745	1.365		"
15	1210	1007	"	"	13.310	1.210		"
16	835	1013	"	"	14.195	1.262		Yellow
17	1100	1010	"	"	14.850	1.660		Reddish yellow
18	1110	1017	"	"	17.205	1.665		Yellow
19	930	1015	Alkaline	"	17.670	3.720	1st week of	Yellowish red
20	1120	1017	Acid	"	22.400	6.720	convalescence.	Yellow, with cloudy deposit.
21	380 <sup>1</sup>	1016	"	"	.....	.....	1st week of	Reddish yellow, with slight cloudy deposit.
22	680	1022	"	"	15.660	4.060	convalescence.	"
23	620	1024	"	"	17.360	6.200		"
24	950	1022	"	"	19.950	9.500		Yellowish-red
25	1480	1020	"	"	22.200	17.760		Reddish-yellow; deposit of lithates.
26	1710	1018	"	"	25.650	20.355		Yellow, with cloudy deposit.
27	2140	1015	"	"	26.840	19.620		Pale yellow, with slight cloudy deposit.
28	1575	1016	"	"	23.050	15.760		Pale
29	1890	1014	"	"	26.460	17.965		Bright
30	830	1020	"	Trace	17.430	7.470		"
31	870	1023	"	"	20.013	10.875		Pale
32	1230	1020	"	None	22.755	14.045		Yellow
33	1300	1017	"	"	23.400	16.400		"
34	1680	1019	"	"	25.200	20.160		Pale yellow
35	1550	1023	"	"	31.000	15.500		Yellow, with slight cloudy deposit.

<sup>1</sup> Some urine lost this day.



Weight on the 22nd day 35·7 kilogrammes or 78·5 lbs. ; on the 38th day 40 kilogrammes or 88 lbs.

The average daily excretion of urinary water, urea, and chlorides during each period has been—

		Amount of water.	Urea.	Chlorides.
		C. C.	Grammes.	Grammes.
1	Before lysis (2 days' urine) .....	·1025	26·302	1·587
	or per kilogramme of body weight .....		·658	·0396
2	During the lysis.....	·1025	16·029	1·354
	or per kilogramme of body weight .....		·400	·0338
3	During 1st week of convalescence .....	·910	17·524	4·002
	or per kilogramme of body weight .....		·433	·100
4	During 2nd week of convalescence .....	1553	22·940	15·472
	or per kilogramme of body weight .....		·523	·3868
5	During 3rd week of convalescence .....	1326	24·473	15·236
	or per kilogramme of body weight .....		·612	·3809

**CASE 11.**—John J—, æt. 30, admitted into the London Fever Hospital March 1st, 1866; is a labourer. Has always had very good health, but lately having been out of work has not lived well. His present illness commenced six days before his admission. He is now, March 1st (seventh day of attack), in a confused and stupid state, not complaining of any pain, rather deaf; his skin is hot and there is a distinct and copious typhus rash visible; his tongue is dry and red; he has been rather sick; he complains of thirst; he has no cough and his lungs are clear to bases: heart sounds are weak but normal.

**Rash.**—Copious, on admission became gradually darker. It began to fade on the twelfth day and was entirely gone by the seventeenth day.

**Sleep.**—Restless, with more or less wandering at night, till the eleventh day. After this he slept well.

**Complexion and Expression.**—Thoroughly typhus, with sordes on lips and teeth till twelfth day, then gradually improved, and was natural on the sixteenth day.

**Tongue.**—Dry, smooth, red, and fissured, till eleventh day,

moist at edges on thirteenth day, slight remains of fissure seen to the twentieth day.

*Bowels*.—Rather confined throughout. On the nineteenth and twenty-ninth days castor oil was taken.

*Headache*.—None after admission.

Nasty taste in mouth much complained of till the eleventh day, then less, only noticed on waking.

*Thirst*.—Intense at first, was complained of more or less till the thirteenth day.

*Appetite*.—Patient complained of feeling hungry on the seventeenth day. His appetite was very good on the twenty-first day.

*Mind*.—Confused, dull, and very deaf till the eleventh day, pretty clear on the fourteenth day; some deafness lasted till the twenty-first day.

*Cough*.—Slight and occasional from the eighth till the fifteenth day. Lungs were clear throughout.

*Heart*.—The impulse could not be felt from the eighth to the thirteenth day. The sounds were normal throughout rather weak at first, especially the first sound.

There was slight desquamation noted from the twenty-first to the thirtieth days.

Patient got up for the first time on the twenty-fifth day. He left the hospital feeling pretty strong and well on the thirty-second day.

His weight, on the twenty-seventh day, was 47·8 kilogrammes, or 104 lbs.; on the thirty-second day was 48·1 kilogrammes, or 106 lbs.

#### *Treatment.*

7th day.   ℞   Mist. Camphoræ ʒj. 4tis horis.  
15th   ,,       Bark mixture three times daily.

#### *Diet.*

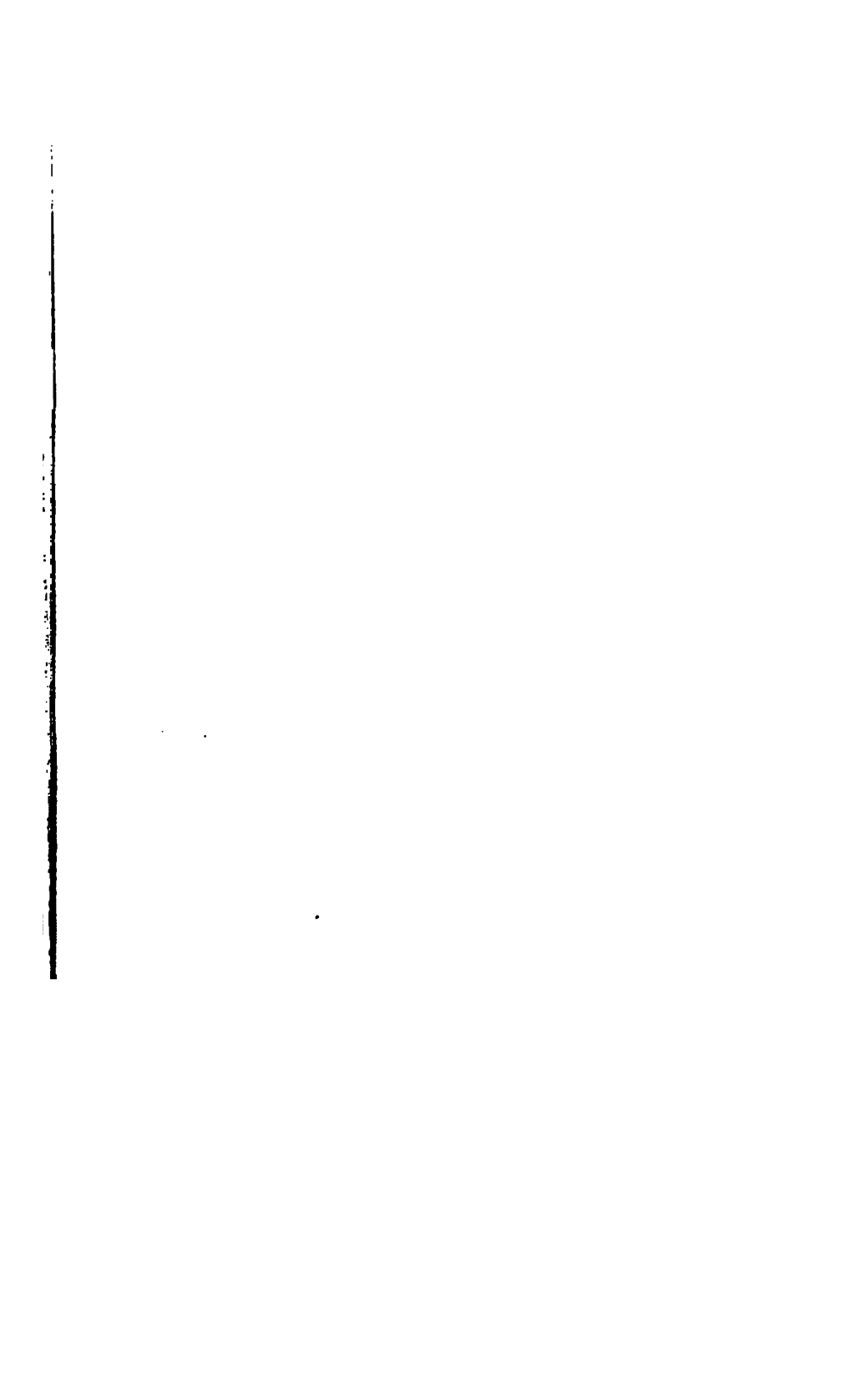
7th day.       Low, beef-tea diet.  
11th   ,,       1 egg, and brandy 4 oz.  
18th   ,,       Fish and middle diet.  
22nd   ,,       Full diet, and porter Oj; omit brandy. (See  
Diagram XI.)

st ;









CASE 11.—John J—, æt. 30. *Analysis of Urine.*

Day of disease.	Quantity of water.	Specific gravity.	Reaction.	Albumen.	Urea.	Chlorides.		Colour and deposit.
	C. C.		Acid	None	Grammes.	Grammes.	Before	
9	720	1015	"	Trace	19.440	.540		Yellowish red; cloudy; no deposit.
10	1210	1015	"	"	27.280	.620		"
11	1110	1016	"	Alb. 1/16 <sup>1</sup>	27.195	.555		Reddish yellow; cloudy.
12	920	1017	"	"				
13	800	1016	"	Less Alb. <sup>2</sup>	22.400	.200		" copious deposit of lithates.
14	1680	1019	"	Less	34.280	None		"
15	470	1020	"	Trace	Some lost	Some lost	During	Reddish yellow
16	1100	1018	"	"	35.200	None		"
17	1270	1017	"	"	31.750	.635		" cloudy.
18	780	1020	"	"	24.180	.585		" lithates.
19	580	1021	"	"	18.560	.580		" lithates.
20	790	1022	"	None	25.280	1.188		Reddish yellow
21	930	1022	"	"	20.460	1.395	1st week of	Yellow; clear.
22	820	1023	"	"	19.680	2.975	convalescence	Reddish yellow; lithates.
23	670	1021	"	"	19.430	3.350		" clear.
24	690	1022	"	"	19.320	4.140		Yellow
25	830	1022	"	"	21.675	5.950		Reddish yellow
26	840	1021	"	"	21.000	5.460		"
27	640	1024	"	"	16.640	2.200		"
28	1550	1016	"	"	26.350	8.525	2nd week of	Yellow
29	1780	1011	"	"	19.580	10.680	convalescence	Bright yellow
30	2160	1011	"	"	21.600	12.960		"
31	2900	1010	"	"	23.200	14.500	3rd wk. of	"
32	1430	1013	"	"	17.160	7.150	conv.	"

<sup>1</sup> The albumen was separated before testing for the urea and chlorides.<sup>2</sup> The albumen not separated, being so little.

Weight on the 27th day 47·3 kilogrammes or 104 lbs.; on the 32nd day 48·1 kilogrammes or 106 lbs.

The average daily excretion of urinary water, urea, and chlorides during each period has been—

		Amount of water.	Urea.	Chlorides.
		C. C.	Grammes.	Grammes.
1	Before lysis (2 days' urine) .....	980	23·300	·580
	or per kilogramme of body weight .....		·485	·012
2	During lysis .....	996	27·958	·378
	or per kilogramme of body weight .....		·581	·007
3	During 1st week of convalescence .....	895	25·015	1·4716
	or per kilogramme of body weight .....		·520	·0305
4	During 2nd week of convalescence .....	1003	20·570	5·758
	or per kilogramme of body weight .....		·427	·119
5	During 3rd week of convalescence .....	2163	20·653	11·536
	or per kilogramme of body weight .....		·430	·239

**CASE 12.**—George Y—, æt. 29, admitted March 7th—fifth day of attack. Patient states that he has been ill for four days, his attack commenced with headache, rigors, and general pains. On admission his mind was quite clear, his skin was hot and there was a copious rather dark rash on trunk and limbs, quite distinct on the dorsum of his feet; the tongue was moist and red at the tip and edges, thickly furred down the centre; the bowels had been opened just before admission; he was breathing quietly and the heart sounds, though weak, were quite normal.

*The Rash.*—Copious on admission grew dark and petechial as the disease advanced. It began to fade on the eleventh day but slight remains of petechiæ were still visible on the eighteenth day.

*Sleep.*—He slept badly and was restless, with a good deal of delirium, till the tenth day, afterwards well.

*Complexion.*—Muddy and flushed, with the suffused and injected eyes and dull look of typhus fever till the tenth day; he then gradually improved and his expression was pretty good on the thirteenth day.







*Tongue*.—Moist and furred at first, grew dry, smooth, and more or less furred, from the eighth to the eleventh days. On the fourteenth it was moist and clean, with slight remains of fissures visible.

*Bowels*.—Open throughout without medicine, were rather loose on the tenth day.

*Thirst*.—Was great till the eighth day, but not complained of after the tenth day.

*Appetite*.—Began to return on the twelfth and was very good by the fifteenth day.

*Cough*.—Never very troublesome. A few bronchitic râles were noted over back from the seventh to the sixteenth days.

*Heart*.—Sounds rather weak, else normal.

Patient was up for the first time on the nineteenth day of his attack. He left at his own request on the twenty-first day. His weight on leaving was 99 lbs, or 45 kilogrammes.

#### *Treatment.*

5th day.	℞	Mist. Camphoræ ʒj. 4tis horis.
6th „		Sedative draught.
7th „		Mustard to back.
10th „		Rep. Mustard back and chest.
14th „		Haust. Cinchonæ ʒj. ter die.

#### *Diet.*

5th day.	Low diet, beef-tea.
6th „	Egg 1, and brandy 4 oz. daily.
10th „	Increase brandy to 6 oz. daily.
14th „	Full diet, with porter Oj; omit brandy. (See Diagram XII.)

CASE 12.—George Y., vol. 16. *Analysis of Urine*

Day of disease	Quantity of water	Specific gravity	Reaction	Albumen	Urea	Chlorides	Color and deposit
7	C C 700	1020	Acid	None	Grammes 21.500	Grammes None	Reddish yellow, copious deposit of lithates
8	Lost	1016	"	Trace	Lost	Lost	
9	800	1017	"	"	28.800	200	cloudy
10	970 <sup>1</sup>	1012	"	None	16.100	486	Yellow
11	370 <sup>1</sup>	1017	"	"	"	"	"
12	900	1019	"	"	20.100	1000	cloudy
13	1320	1013	"	"	25.000	2400	cloudy
14	1450	1011	"	"	21.750	2400	"
15	1310	1012	"	"	22.925	3080	"
16	1680	1010	"	"	22.080	6720	Reddish yellow, cloudy
17	1860	1011	"	"	13.000	6020	"
18	1810	1014	"	"	21.720	6385	"
19	1280 <sup>1</sup>	1016	"	"	"	3525	Bright yellow
20	1410	1010	"	"	16.920	"	"
21	1490	1012	"	"	16.370	2206	rather cloudy

<sup>1</sup> Some urine lost on these days.

Weight on the 21st day 45 kilogrammes or 99 lbs.

The average daily excretion of urinary water, urea, and chlorides during each period has been—

		Amount of water.	Urea.	Chlorides.
		C. C.	Grammes.	Grammes.
1	Before lysis (1 day's urine) ..... or per kilogramme of body weight .....	·700	24·500 ·544	None
2	During lysis (1 day's urine)..... or per kilogramme of body weight .....	·800	28·800 ·640	·200 ·0044
3	During 1st week of convalescence ..... or per kilogramme of body weight .....	·1404	21·992 ·489	4·206 ·0934
4	During 2nd week of convalescence ..... or per kilogramme of body weight .....	·1450	18·145 ·403	2·830 ·063

**CASE 13.**—Edward S—, æt. 29, admitted into the London Fever Hospital, October 9th, 1865. Has been ill six days. He is a railway porter and is a strong healthy-looking man, he has always had good health, but lately has been out of work and not living very well. On admission, seventh day of his attack, his skin was hot with a typhus rash well out; his tongue was moist and furred and his bowels had been freely opened by medicine. He was complaining much of headache and general pains; sleeps badly and has wandered a little at night before admission.

**Rash.**—Well out on admission, was never very copious or dark. It began to fade on the thirteenth day and was gone on the fifteenth day.

**Sleep.**—Fair throughout attack; little wandering on eighth, ninth, and tenth, days.

**Complexion.**—Dusky and flushed, with suffused and injected eyes and heavy vacant expression till the eleventh day, was quite clear and natural on the fourteenth day. No sordes on lips throughout.

**Tongue.**—Moist and covered with a thick yellowish creamy fur till twelfth day, was less furred on thirteenth, and clean on the sixteenth day.

*Bowels.*—Inclined to be loose at first were open daily throughout attack.

*Thirst.*—Much complained of till the ninth day.

*Appetite.*—Patient complained of feeling hungry on the fourteenth day, but his appetite was not good till the eighteenth day.

*Cough.*—Rather troublesome occasionally.

*Lungs.*—Respiration over back rather harsh with some sibilant râles audible ninth, tenth, and eleventh days. No dulness on percussion.

*Heart.*—Sounds normal throughout; first sound rather weak, but impulse was felt distinctly throughout.

*Perspiration.*—Patient perspired freely on nights of thirteenth and fourteenth, and numerous sudamina were seen on chest and abdomen.

Patient was up for the first time on the sixteenth day for a few hours; he left the hospital quite well and strong on the thirty-fourth day.

Patients weight on the nineteenth day was 57 kilogrammes, or 125·5 lbs; on the thirty-fourth day was 70·7 kilogrammes, or 155·5 lbs.

#### *Treatment.*

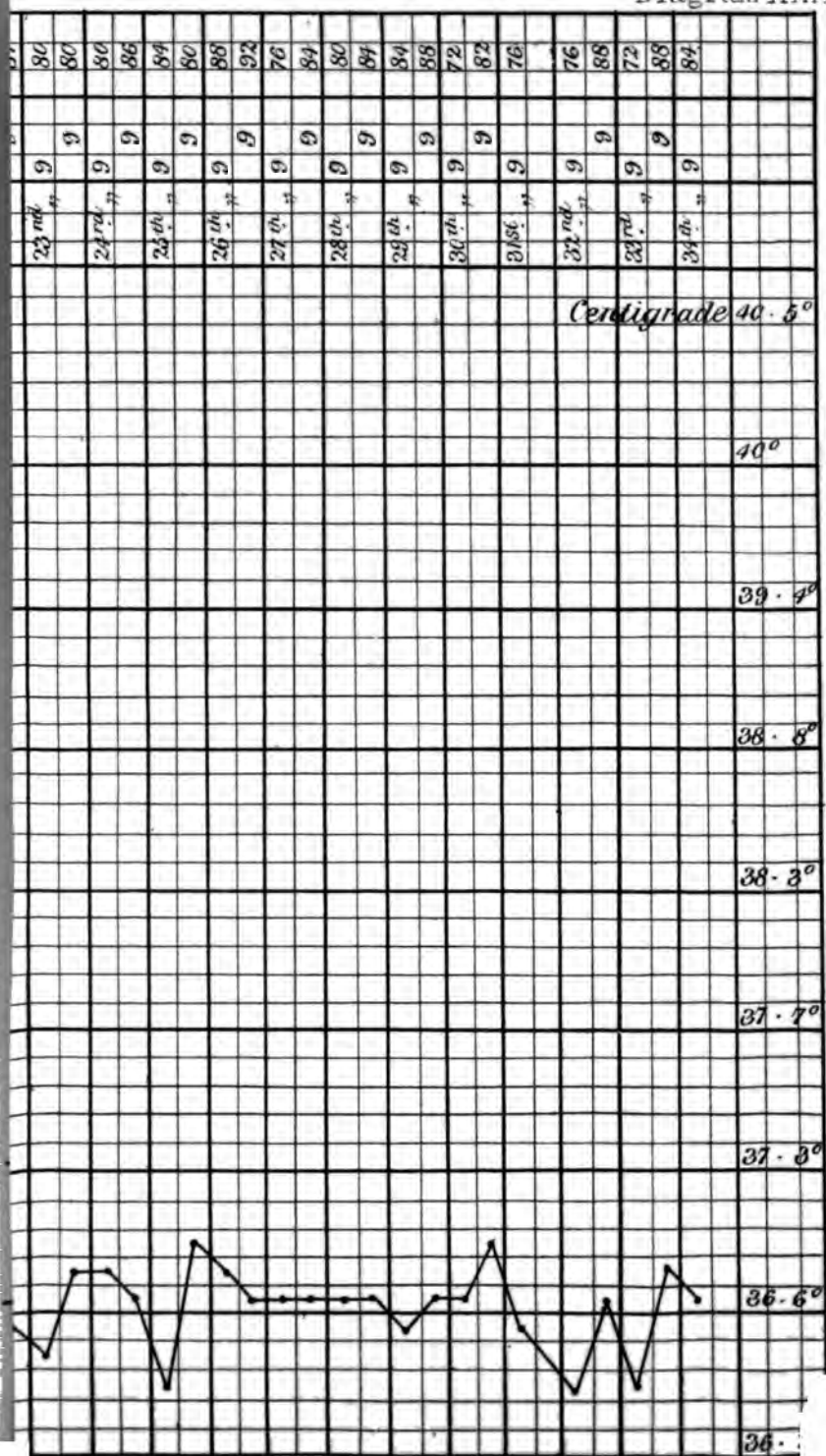
7th day.	R	Æther mixture ʒj, 4tis horis.
14th „		Bark mixture ʒj, ter die.

#### *Diet.*

7th day.	Low diet, with beef-tea.
14th „	Full diet, and porter Oj.
18th „	1 egg. (See Diagram XIII.)

day of his Illness.

Diagram XIII.







CASE 13.—Edward S—, æt. 29. *Analysis of Urine.*

Day of disease.	Quantity of water.	Specific gravity.	Reaction.	Albumen.	Urea.	Chlorides.		Colour and deposit.
	C. C.		Acid	None	Grammes.	Grammes.	During lysis	
9	1600	1015	"	"	36.750			
10	1875	1015	"	"	50.625			
11	1680	1015	"	"	46.200			
12	1540	1015	"	"	43.880			
13	1150	1015	"	"	38.350	.287		Pale yellow; uric acid deposit.
14	1050	1016	"	"	31.500	2.100		" "
15	960	1019	"	"	27.360	2.700		" "
16	905	1020	"	"	23.530	4.977		" "
17	1480	1011	"	"	27.180	6.660		" "
18	1330	1018	"	"	25.935	10.640		" "
19	1830	1015	"	"	23.790	11.895		" "
20	2200	1013	"	"	24.200	16.500		less uric acid deposit.
21	2135	1010	"	"	26.687	16.012		cloudy.
22	2185	1014	"	"	24.035	17.480		less uric acid deposit.
23	1090	1016	"	"	17.440	10.082		slight cloudy deposit.
24	1300	1018	"	"	24.700	14.950		Bright yellow "
25	1510	1011	"	"	28.690	15.100		" "
26	1920	1019	"	"	37.440	19.200		no deposit.
27	1825	1017	"	"	31.025	16.425		" "
28	2530	1017	"	"	32.430	18.270		slight cloudy deposit.
29	2380	1011	"	"	36.890	22.210		Pale yellow; no deposit.
30	2510	1010	"	"	47.690	25.210		" "
31	1720	1020	"	"	36.980	18.920		Bright yellow; slight cloudy deposit.
32	2200	1019	"	"	39.600	20.900		Pale yellow; no deposit.
33	1890	1017	"	"	35.910	15.985		" "
34	2740	1017	"	"	44.580	23.280		Bright yellow "

Weight on the 19th day 57 kilogrammes or 125·5 lbs. ; on the 34th day 70·7 kilogrammes or 155·5 lbs.

The average daily excretion of urinary water, urea, and chlorides for each period has been—

	Amount of water.	Urea.	Chlorides.
	C. C.	Grammes.	Grammes.
1 Before lysis. No urine obtained.			
2 During lysis ..... or per kilogramme of body weight .....	·1648	44·366 ·627	
3 During 1st week of convalescence ..... or per kilogramme of body weight .....	·1243	27·521 ·389	6·376 ·0901
4 During 2nd week of convalescence ..... or per kilogramme of body weight .....	·1763	25·599 ·362	15·617 ·221
5 During 3rd week of convalescence ..... or per kilogramme of body weight .....	·2162	34·144 ·539	20·026 ·283

CASE 14.—Henry D—, æt. 22, admitted into the London Fever Hospital October 18th, 1866, costermonger ; single. Present illness commenced eight days ago with headache, general pains, and rigors. His friends say that he has been very delirious. On admission his skin was hot with a distinct, but not copious, typhus rash. He did not complain of any pain ; was lying on his back with eyes closed and breathing quietly ; was rather stupid but answered questions when roused ; his tongue was moist at edges, dryish and rough down centre ; the bowels had been open before admission ; heart sounds weak but normal, and the lungs were clear at bases.

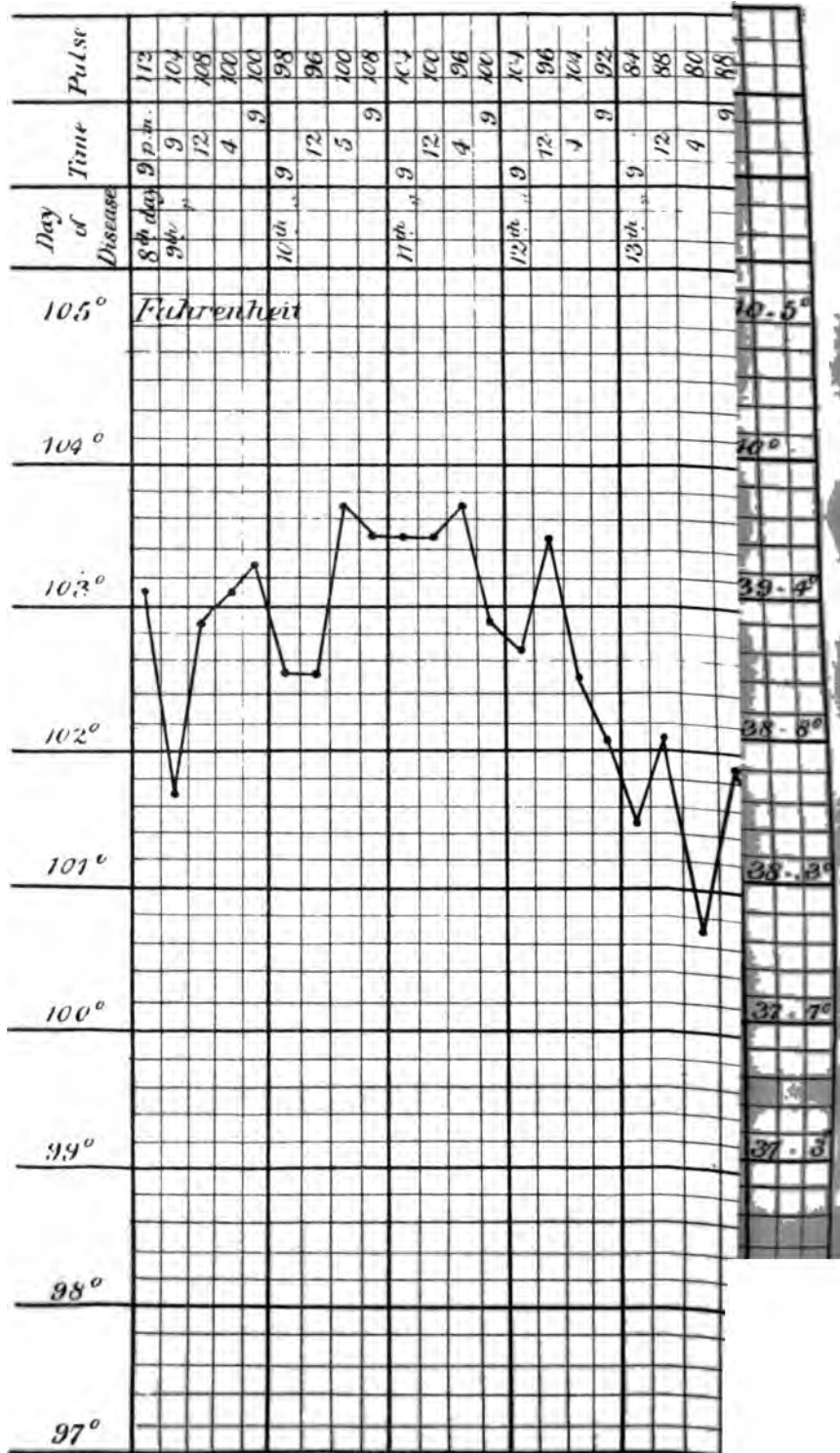
*Rash.*—Distinct but not very copious, was noted as fading on the eleventh day, and was entirely gone by the sixteenth day.

*Sleep.*—Disturbed with a good deal of wandering till the eleventh, then slept well.

*Complexion.*—Muddy and sallow with suffused and injected eyes, and dull vacant expression till the twelfth day, clear and natural on the sixteenth day.







*Tongue*.—Dry and rough down centre on admission, was brown and cracked slightly eleventh and twelfth days, then began to improve, and on the sixteenth day was moist and clean.

*Bowels*.—Rather loose at first, were open throughout acute stage without medicine; rather confined during convalescence.

*Thirst*.—Much complained of till eleventh day, not after.

*Appetite*.—Returning on the fifteenth day, good on seventeenth day.

*Mind*.—Confused and dull till twelfth day, then improved and was quite clear by sixteenth day.

*Cough*.—Rather troublesome from the ninth to sixteenth days.

*Lungs*.—Few sibilant râles audible over back till the seventeenth day.

*Heart*.—Sounds normal throughout, rather weak at first.

Patient was up for the first time on the twenty-fifth day. He left the hospital feeling well and strong on the thirty-third day.

His weight on the twenty-fifth day was 48·4 kilogrammes, or 106·5 lbs.; on the thirty-third day was 51·8 kilogrammes, or 114 lbs.

#### *Treatment.*

8th day.	℞	Mist. Camphoræ ʒj, 4tis horis.
15th	„	Back mixture three times daily.
23rd	„	Castor oil ʒj, at once.

#### *Diet.*

8th day.	Low diet with beef-tea.
15th	„ Full diet, porter Oj.
17th	„ Not to have porter.
24th	„ 1 egg. (Sec Diagram XIV.)

CASE 14.—Henry D—, æt. 22. *Analysis of Urine.*

Day of disease.	Quantity of water.	Specific gravity.	Reaction.	Albumen.	Urea.	Chlorides.		Colour and deposit.
	C. C.				Grammes.	Grammes.		
11	Lost	1020	Acid	Trace	25.080	1.045	Before	Pale yellow; cloudy.
12	1045	1017	"	Less	23.780	.610	lysis	deposit of lithates.
13	1220	1020	"	None				"
14	1900	1015	Alkaline	"	50.350	.950	Dur.	cloudy; copious deposit of lithates.
15	2010	1011	Acid	"	42.220	2.010		no deposit.
16	1760	1016	"	"	48.500	1.760		Reddish yellow "
17	1080	1018	"	"	33.980	1.545		cloudy deposit.
18	1080	1027	"	"	36.555	4.120		Yellowish red; deposit of lithates.
19	1010	1026	"	"	33.633	5.555		Reddish yellow "
20	1040	1021	"	"	32.760	5.720		"
21	930	1024	"	"	30.680	3.720		Yellowish-red; cloudy deposit.
22	870	1021	"	"	25.230	2.175		Reddish yellow; no deposit.
23	670	1017	"	"	21.105	2.345	1st week of convalescence	slight deposit of lithates.
24	665	1028	"	"	24.937	4.332		cloudy deposit; few phosphates.
25	500	1025	"	"	18.500	2.250		deposit of lithates.
26	1240	1023	"	"	39.060	9.300	2nd week of convalescence	Yellow; cloudy deposit.
27	910	1020	"	"	26.845	5.915		Reddish yellow; cloudy deposit.
28	1435	1019	"	"	29.925	12.635		Bright yellow; slight cloudy deposit.
29	1560	1016	"	"	24.960	13.260		"
30	980	1020	"	"	17.640	9.555		Pale yellow "
31	800	1020	"	"	15.200	9.600	3rd week of convalescence	Bright yellow "
32	1970	1023	"	"	25.400	15.875		Yellow "
33	960	1023	"	"	21.600	12.480		"

Weight on the 25th day 48·4 kilogrammes or 106·5 lbs.; on the 33rd day 51·8 kilogrammes or 114 lbs.

The average daily excretion of urinary water, urea, and chlorides during each period has been—

		Amount of water.	Urea.	Chlorides.
		C. C.	Grammes.	Grammes.
1	Before lysis (2 days' urine) .....	1132	24·435	·8275
	or per kilogramme of body weight .....		·473	·0159
2	During lysis .....	1890	47·023	1·573
	or per kilogramme of body weight .....		·907	·0303
3	During 1st week of convalescence .....	940	30·567	3·594
	or per kilogramme of body weight .....		·590	·0695
4	During 2nd week of convalescence .....	1040	25·923	8·405
	or per kilogramme of body weight .....		·500	·162
5	During 3rd week of convalescence .....	1010	20·733	12·651
	or per kilogramme of body weight .....		·400	·244

CASE 15.—Joseph A—, æt. 20, shoemaker, admitted into the London Fever Hospital, November 10th. Attack commenced yesterday (November 9th) whilst at work, with headache, pains in back and limbs, and rigors. Patient felt quite well the day before yesterday. Has always had good health. Body is well nourished.

On admission was suffering much from headache and general pains; his face was flushed; the skin hot, but there was not any distinct rash. The tongue was moist and furred; the bowels had been opened the day before. He was very thirsty, and rather restless. Lungs and heart were quite healthy. Mind quite clear.

*Rash.*—On the third day the skin of trunk was injected and a few rose spots, slightly elevated, were to be seen here and there; on the fourth day there was a distinct typhus rash on the forearms, and backs of hands, and on the fifth day it was quite distinct on the trunk; never very petechial, but noted as dark and copious during the course of the disease. It began to fade on the twelfth, and was quite gone by the fifteenth day.



*Sleep*.—His sleep was restless and disturbed, with much delirium from the fifth to the tenth day, then gradually improved. On the fourteenth slept well, and continued to do so.

*Complexion*.—Dusky and flushed, with suffused and injected eyes, dry lips, but no sordes throughout, and a heavy listless vacant expression till the fifteenth day. Noted as clear, but rather pallid, with good expression, on the nineteenth day.

*Tongue*.—On the eleventh and twelfth days it was noted as dry and brown down the centre, and again slightly so on the fourteenth and fifteenth days, at other times it was moist and furred. Not noted as quite clean till the twenty-first day.

*Bowels*.—Were open throughout, without medicine; rather loose from the sixth to the ninth days.

*Headache* and general pains, with nasty taste in mouth, were complained of more or less till the eighth day. Headache very bad indeed the first four days.

*Appetite*.—First complained of hunger on the fourteenth, but the appetite was not good till the twentieth day.

*Cough*.—Slight, with some little frothy expectoration, from the sixth to the fourteenth days.

*Heart* sounds on admission were weak, but otherwise normal. From the twelfth to the nineteenth days, a soft blowing systolic murmur was audible at the mitral apex. On the twenty-fifth day it had quite gone and was not heard afterwards.

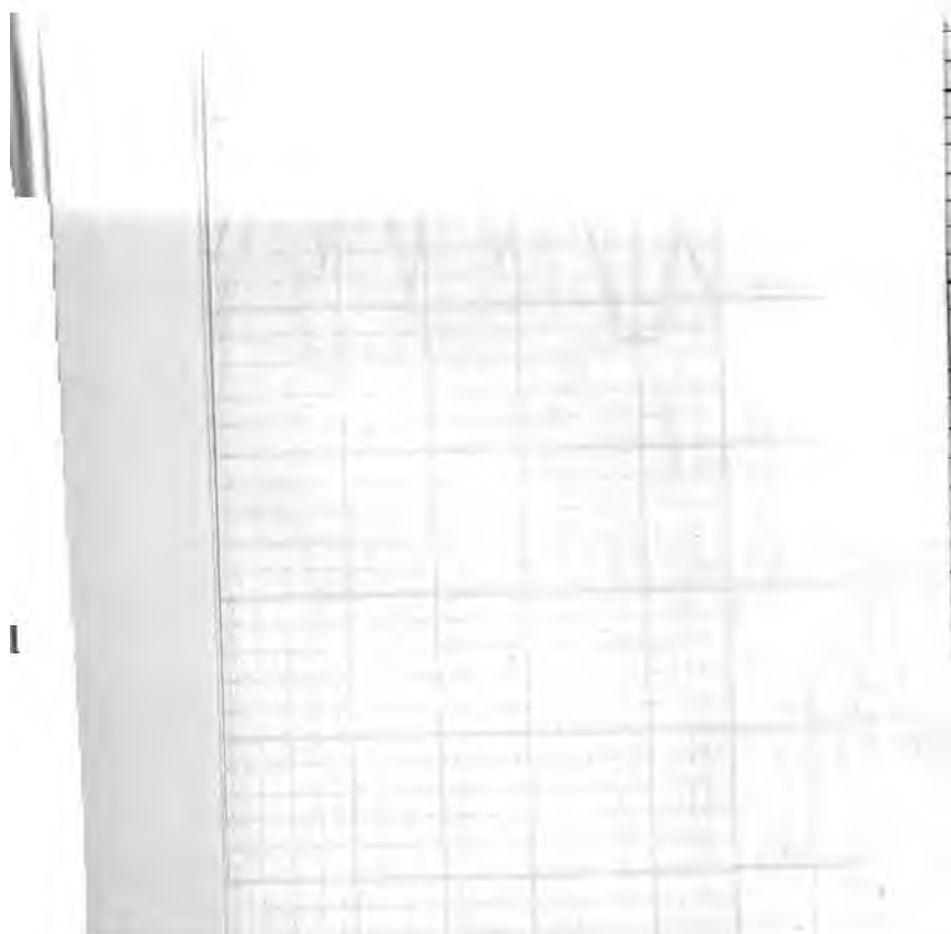
Patient did not get up till the twenty-sixth day, having to wait for his clothes, then felt very well and strong. He left the Hospital on the thirtieth day of his illness, at his own request, said he felt strong enough to work.

Weight on twenty-sixth day 51·1 kilogrammes, or 112·5 lbs.; on thirtieth day 52·3 kilogrammes, or 115½ lbs.

#### *Treatment.*

2nd day. ℞ Dilute Hydrochloric acid ℥xv, dilute Nitric acid ℥xv, Spirit of Nitrous ether ʒss, Syrup ʒss, water to ʒj. Mixture to be taken every four hours.





10th day. Linseed and mustard poultice to loins.  
19th „ Sulphate of Quinine gr. ij, dilute Sulphuric  
Acid mix, water ʒj. To be taken three  
times daily.

*Diet.*

2nd day. Low beef tea diet.  
19th „ Wine 4 oz., egg one.  
20th „ Full diet and porter Oj. To leave off wine.  
(See Diagram XV.)

Case 15.—Joseph A—, æt. 20. *Analysis of Urine.*

Day of disease.	Quantity of water.	Specific gravity.	Reaction.	Albumen.	Urea.	Chlorides.	Before crisis	During crisis	1st 5 days of convalescence	2nd week of convalescence	Colour and deposit.
	C. C.				Grammes.	Grammes.					
5	1310	1028	Acid	None	55.200	3.595	Yellowish red; slight cloudy deposit.				Yellowish red; slight cloudy deposit.
6	820	1030	"	"	35.670	2.870	"				" lithates.
7	880	1028	"	Slight amount	.....	.....	" no deposit.				"
8	880	1025	"	"	.....	.....	"				"
9	1500	1018	"	More	45.000	Trace	"				" alb. separated before ex- aminating for urea.
10	1180	1022	"	"	44.635	.565	"				"
11	830	1022	"	"	32.370	Trace	"				"
12	1660	1021	"	½ part	65.570	"	Reddish yellow				"
13	1390	1016	"	½ "	48.650	"	" lithates				"
14	1050	1020	"	½ "	42.000	"	"				"
15	1250	1021	"	None	51.250	.625	" copious				"
16	1830	1017	Alkaline	"	61.305	1.930	" cloudy.				"
17	1490	1015	"	"	32.780	3.725	Yellow; copious deposit of lithates.				"
18	1460	1017	"	"	30.660	5.840	"				"
19	1630	1016	Acid	"	30.970	7.335	Bright yellow; muddy.				"
20	1250	1020	"	"	32.520	2.500	Reddish yellow; slight cloudy deposit.				"
21	1440	1016	"	"	34.560	2.160	"				"
22	1370	1015	"	"	26.715	2.055	" lithates.				"
23	1310	1013	"	"	23.580	3.275	"				"
24	1600	1010	"	"	24.000	4.800	" cloudy deposit.				"
25	1520	1014	"	"	25.080	4.360	"				"
26	1540	1013	"	"	23.100	5.390	Yellow; slight				"
27	1790	1011	"	"	21.480	4.475	Reddish yellow; cloudy; no deposit.				" phosphates.
28	1650	1012	"	"	24.800	5.425	" deposit of lithates.				"
29	970	1015	"	"	17.460	5.225	"				"
30	2100	1011	"	"	21.000	11.550	Yellow; cloudy; phosphates.				"

Weight on the 26th day 51·1 kilogrammes or 112·5 lbs.; on the 30th day 52·3 kilogrammes or 115·5 lbs.

The average daily excretion of urinary water, urea, and chlorides during each period has been—

		Amount of water.	Urea.	Chlorides.
		C. C.	Grammes.	Grammes.
1	Before lysis (5 days' urine) .....	1060	42·575	1·406
	or per kilogramme of body weight .....	.....	·814	·0268
2	During the lysis.....	1445	50·259	1·030
	or per kilogramme of body weight .....	.....	·961	·0197
3	During 1st 5 days' of convalescence, the temperature being high .....	1430	31·085	3·978
	or per kilogramme of body weight .....	.....	·961	·0760
4	During 8 succeeding days, the temperature being still high .....	1622	22·562	5·563
	or per kilogramme of body weight .....	.....	·421	·106

CASE 16.—James H—, æt. 16, admitted into the London Fever Hospital, October 12th 1865, was born and has lived in London all his life. Is a paper-hanger by trade. He has always lived pretty well, and has had very good health.

His present attack commenced six days before his admission, with headache, rigor and general pains. When first seen (October 12th) his headache had entirely left him, he complained of much thirst; his skin was very hot and covered with fleabites, and there was a faint typhus rash visible.

*Rash.*—Distinct on admission, grew darker as the disease advanced. It was never very copious. It began to fade on the thirteenth day, and was quite gone by the sixteenth day.

*Sleep.*—He slept pretty well throughout, restless but not wandering except on the ninth.

*Complexion.*—Dusky and flushed, with suffused and injected eyes and slight sordes on lips and teeth till the eleventh day, then gradually improved, and on the sixteenth day it was noted *clear*. His expression, at first vacant and dull, was good on the sixteenth day.

*Tongue.*—With the exception of the eleventh day, when it

was dryish at tip and down centre, the tongue was moist and furred throughout. It was quite clear on the fifteenth day.

*Bowels.*—Were open throughout without medicine, were rather loose on the eighth. Natural stool on the sixteenth day.

*Thirst.*—Much complained of till tenth day, but not afterwards.

*Appetite.*—Returning on the twelfth day. Good on the sixteenth day.

*Mind.*—Confused and dull to the thirteenth day, then improved; and on the sixteenth day was quite clear.

*Lungs.*—Cough nothing to speak of throughout, no dulness chest or back. Respiration noted as rather harsh at bases from the ninth to the fourteenth days.

*Heart.*—Sounds and dulness normal throughout.

Patient was up for about two hours on the sixteenth day. He left the hospital on the twenty-third day, feeling quite well and strong.

Weight on the sixteenth day 38·2 kilogrammes, or 84 lbs.

„ twenty-third „ 41·8 „ or 92 lbs.

#### *Treatment.*

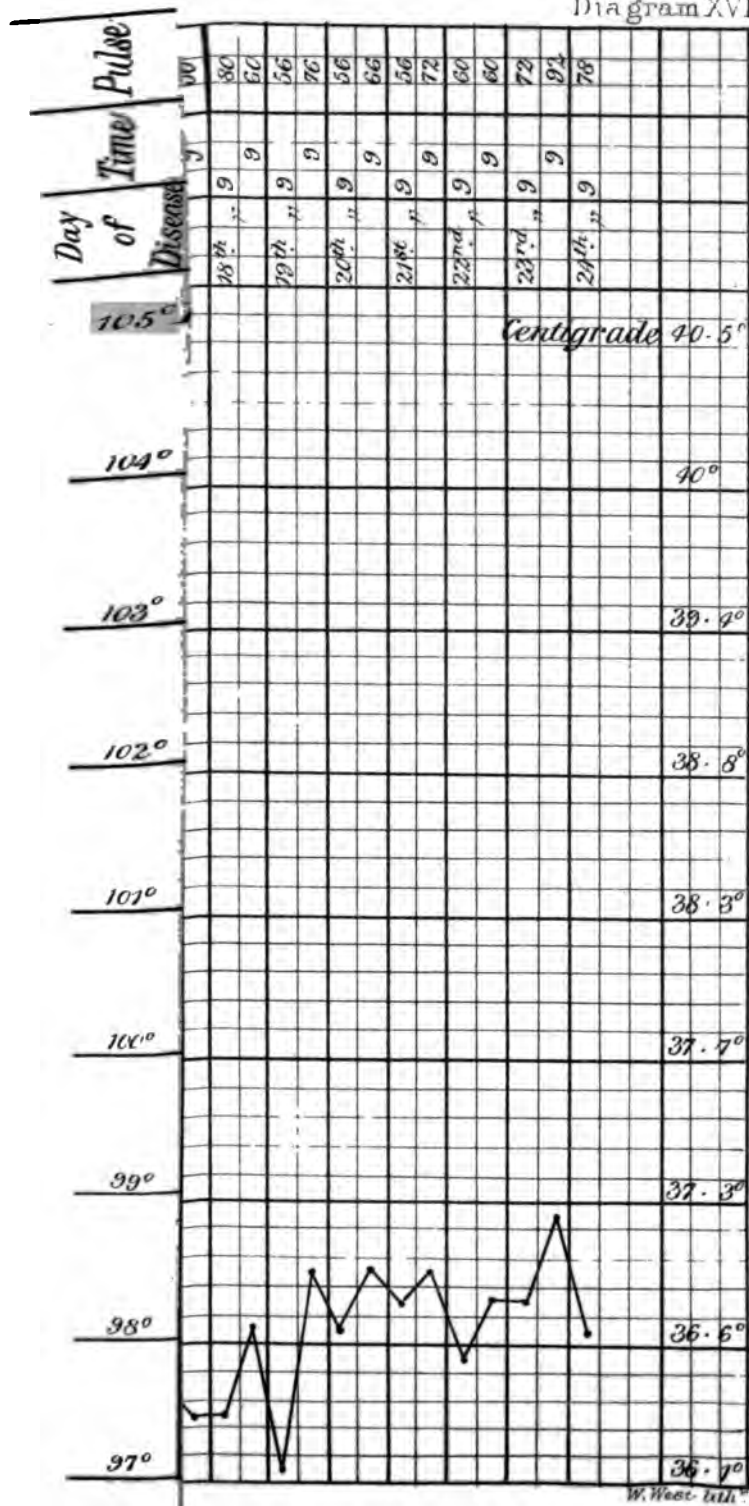
7th day.	R	Nitric acid mixture every four hours.
8th „		Chalk of catechu mixture occasionally.
10th „		Bark mixture three times daily.

#### *Diet.*

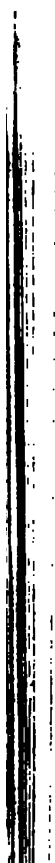
7th day.	R	Low, beef-tea diet.
14th „		Full diet. (See Diagram XVI.)

p. 402' day of his illness.

Diagram XVI







CASE 16.—Joseph H—, æt. 16. *Analysis of Urine.*

Day of disease.	Quantity of water.	Specific gravity.	Reaction.	Albumen.	Urea. Grammes. 22·500	Chlorides. Grammes. .....	Before		Colour and deposit.
							lys.	lys.	
9	C. C. 1500	1010	Acid	None					
10	2010	1008	"	"	32·160	1·005			
11	1940	1009	"	"	30·070	·495			
12	Lost	1010	"	"					
13	1550	1010	"	"	29·450	·775			
14	1070	1009	"	"	17·120	·635			
15	720	1010	"	"	15·840	·540			
16	955	1006	"	"	12·415	·477			
17	1330	1012	"	"	22·610	·987			
18	1250	1011	"	"	21·250	1·250			
19	1240	1010	Slightly Alkaline	"	19·840	3·720			Pale yellow; slight deposit of lithæa.
20	1050	1010	"	"	20·475	3·675			" copious
21	1140	1017	"	"	24·510	6·270			Yellow
22	1390	1013	"	"	24·230	9·730			"
23	880	1017	"	"	19·360	4·840			"
24	1380	1017	Acid	"	20·010	12·420			Bright yellow; no deposit.

Weight on the 16th day 38·2 kilogrammes or 84 lbs. ; on the 23rd day 41·8 kilogrammes or 92 lbs.

The average daily excretion of urinary water, urea, and chlorides has been during each period—

		Amount of water.	Urea.	Chlorides.
		C. C.	Grammes.	Grammes.
1	Before lysis (1 day's urine) .....	1500	22·500	.....
	or per kilogramme of body weight .....	.....	·538	.....
2	During lysis .....	1642	27·200	·7025
	or per kilogramme of body weight .....	.....	·651	·0168
3	During 1st week of convalescence .....	1240	19·563	2·417
	or per kilogramme of body weight .....	.....	·468	·0578
4	During 2nd week of convalescence .....	1216	21·200	8·996
	or per kilogramme of body weight .....	.....	·505	·215

CASE 17.—Henry C—, æt. 15, labourer, admitted into the London Fever Hospital, March 1st, 1866.

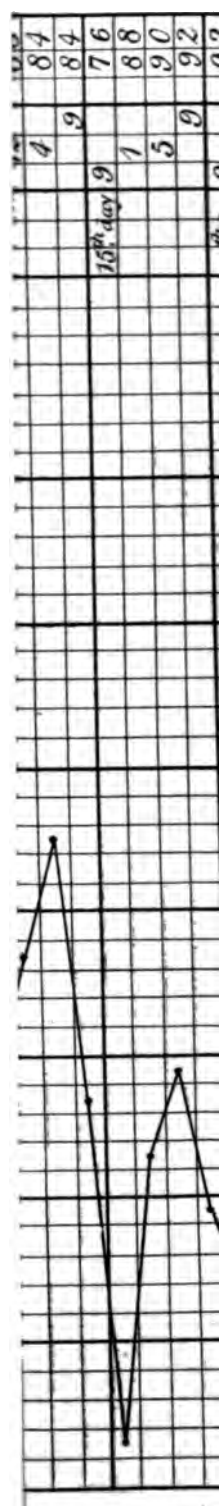
Patient states that he has been ill four days. His attack commenced with headache and general pains. On admission his mind was quite clear, he answered questions readily—his skin was hot, and there was a distinct though not copious typhus rash to be seen. The tongue was moist and thinly furred down centre, red at tip and edges. The bowels had been opened the day before—he had no cough, but he complained much of headache and general pains.

*The rash.*—Distinct on admission, was not very copious throughout the attack ; it gradually grew darker and began to fade on the twelfth day, and was entirely gone by the sixteenth day.

*Sleep.*—He slept pretty well throughout ; he wandered a little on the nights of the tenth and eleventh days, and on the afternoon of the fifteenth day.

*Complexion.*—Dusky and flushed with suffused and injected eye, dry lips up to the eleventh day, then gradually improved, and was noted as clear with good expression on the eighteenth day.





*Tongue*.—Furred throughout, was rather dry at the tip till the eighth day. Clean and moist on seventeenth day.

*Bowels*.—Were open throughout without medicine.

*Headache and general pains*.—Rather severe at first, disappeared after the ninth day.

*Thirst*.—Much complained of till the tenth day.

*Appetite*.—Returning on the thirteenth, good on the sixteenth day.

*Mind*.—No great wandering throughout, but mind was very confused from the ninth till the eleventh day.

*Cough*.—Not complained of; lungs were clear throughout.

*Heart*.—Sounds weak—during the second week of attack, else normal.

Slight desquamation of the skin was noted from the nineteenth to the twenty-first days.

Patient got up for the first time on the twenty-second day. On the twenty-fifth day he weighed 75·5 lbs. or 34·3 kilogrammes.

On the thirty-sixth day he weighed 84·5 lbs. or 38·4 kilogrammes.

#### *Treatment.*

5th day of attack.	R	Mist. Camphoræ, ʒj, 4tis horis.
14th     "       "		Haust. Cinchonæ, ʒj, t. d. s.

#### *Diet.*

5th day.	Low, beef-tea diet.
14th     "	Middle diet and fish.
17th     "	Middle diet and chops. (See Diagram XVII.)

CASE 17.—Henry C—, æt. 15. *Analysis of Urine.*

Day of disease.	Quantity of water.	Specific gravity.	Reaction.	Albumen.	Urea.	Chlorides.		Colour and deposit.
	C. C.				Grammes.	Grammes.		
7	1430	1013	Acid	None	22·880	1·430	Before 1 yrs.	Yellow; slight cloudy deposit.
8	1140	1017	"	"	17·670	·885		" clear.
9	960	1017	"	"	15·360	·480		" "
10	500 <sup>1</sup>	1019	"	"	15·900	·530		Yellow; copious deposit of lithates.
11	1060	1017	"	"	15·180	None		" cloudy.
12	660	1023	"	"	14·000	"		" lithates.
13	400	1023	"	"	26·040	·465		Reddish yellow; cloudy; lithates.
14	930	1017	"	"	27·600	·690	1 yrs.	Yellow; clear.
15	1380	1013	"	Trace	29·520	·615		Reddish yellow; clear.
16	1230	1016	"	"	13·340 <sup>1</sup>	·580 <sup>1</sup>	1st week of convalescence.	Yellow; clear.
17	580 <sup>1</sup>	1018	"	None	18·690	·885		" "
18	590	1023	"	"	17·280	2·520		" "
19	640	1021	"	"	17·825	9·875		" "
20	775	1018	"	"	17·820	4·950		Reddish yellow; clear.
21	660	1022	"	"	13·160	4·480		" "
22	560	1020	"	"	13·800	4·500		" phosphates.
23	600	1022	"	"	15·120	5·040		" "
24	630	1022	"	"	19·140	7·830		Reddish yellow; cloudy; lithates.
25	870	1023	"	"	17·480	10·160		Yellow; slight deposit.
26	1270	1016	"	"	16·200	6·480		" clear.
27	1080	1017	"	"	23·290	12·330		Bright yellow; clear.
28	1370	1013	"	"	21·110	14·770		" "
29	2110	1012	"	"	20·040	9·185	2nd week of convalescence.	" "
30	1670	1013	"	"	22·605	10·960		Yellow
31	1370	1017	"	"	27·600	13·800	3rd week of convalescence.	Bright yellow
32	2300	1011	"	"	27·600	5·320		"
33	1380	1015	"	"	21·945	18·400		Yellow
34	2300	1015	"	"	34·500			Bright yellow

! Some lost on each of these days.

Weight on the 25th day 34·8 kilogrammes or 75·5 lbs. ; on the 36th day 38·4 kilogrammes or 84·5 lbs.

The average daily excretion of urinary water, urea, and chlorides during each period has been—

		Amount of water.	Urea.	Chlorides.
		C. C.	Grammes.	Grammes.
1	Before lysis (6 days' urine) .....	941	16·831	·552
	or per kilogramme of body weight .....	.....	·438	·0143
2	During lysis .....	1180	27·720	·590
	or per kilogramme of body weight .....	.....	·722	·0153
3	During 1st week of convalescence .....	637	16·429	3·535
	or per kilogramme of body weight .....	.....	·228	·0920
4	During 2nd week of convalescence .....	1285	18·911	9·399
	or per kilogramme of body weight .....	.....	·492	·242
5	During 3rd week of convalescence .....	1825	26·662	12·120
	or per kilogramme of body weight .....	.....	·794	·318

CASE 18.—Edmund J—, æt. 11 years, admitted into the London Fever Hospital, January 1st, 1866. Father labourer.

Says that he first felt ill yesterday (December 30th), the day before was running about and quite well. His attack commenced with rigors, headache, and pains all over.

On admission his skin was hot, somewhat injected, but there was no distinct rash, numerous fleabites. His tongue was moist and red at tip and edges with thin white fur on dorsum. He complained of much thirst and headache; he had slight cough, and there were a few sonorous and sibilant râles audible over the back, but no dulness. The heart-sounds were quite healthy and his mind quite clear.

*Rash.*—There was no distinct typhus rash throughout, but on the third, fourth, and fifth days pinkish mottling was noted on the arms and trunk.

*Sleep.*—He slept well throughout; profuse perspiration was noted on the fifth and slight on the seventh and eighth. No delirium at all.

*Complexion.*—Slightly flushed from the third to the fifth



day. Expression noted as rather listless on the fourth, fifth, and sixth days; good on the seventh.

*Tongue.*—Noted as moist throughout; thinly furred up to the eighth day.

*Bowels.*—Open throughout without medicine.

*Headache with general pains.* Rather severe to the fourth day; nasty taste in his mouth complained of till the seventh day; never at all deaf.

*Appetite.*—First complained of being hungry on the sixth day; appetite was noted as good on eleventh day.

*Cough.*—Slight throughout; no expectoration.

*Lungs.*—Few sonorous râles were audible over chest the first seven days.

*Heart.*—Sounds normal throughout, with the exception of slight reduplication of the second sound, first noted on the fourth day.

Patient got up for the first time on the fourteenth day, and was discharged well and strong on the twenty-second day.

Patient weighed on the fourteenth day 55·5 lbs., or nearly 25·23 kilogrammes.

### *Treatment.*

2nd day. R Mist. Camphoræ ʒj, every four hours.

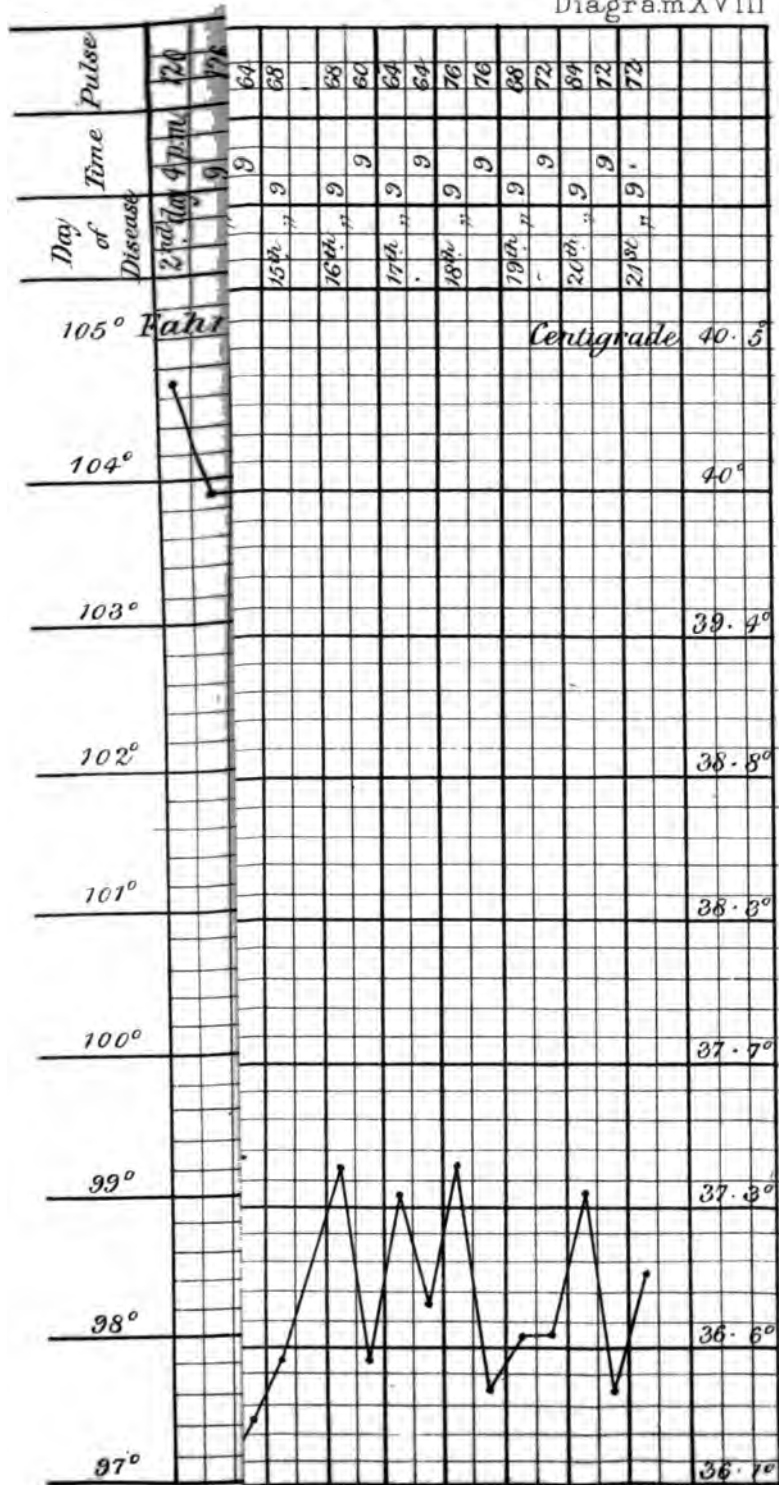
9th „ Bark mixture ʒss, three times daily. Mustard to back.

### *Diet.*

2nd day. Low, beef-tea.

9th Fish, and middle diet. (See Diagram XVIII.)

Diagram XVIII





CASE 18.—Edmund J—, æt. 11. *Analysis of Urine.*

Day of disease.	Quantity of water.		Specific gravity.	Reaction.	Albumen.	Urea.		Chlorides.			Colour and deposit.
	C. C.	oz.				Grammes.	Grains.	Grammes.	Grains.		
4	1080	38	1017	Acid	None	32.940	507.2	1.620	24.9	Before	Yellowish red; clear.
5	1240	43½	1012	"	"	26.040	401	1.240	19.0	lys.	"
6	820	28½	1015	"	"	20.500	315.7	.820	12.5	lys.	Yellow
7	Lost	Lost	1017	"	"	.....	.....	.....	.....		"
8	650	22½	1015	"	"	13.650	209.2	.650	10.0		Reddish yellow
9	450	15½	1017	"	"	10.350	159.3	.900	13.8		Yellowish red
10	640	22½	1019	"	"	12.800	197.1	4.160	64		Yellow; cloudy deposit; phosphates.
11	620	21½	1015	"	"	11.780	181.4	5.890	90.7		" clear.
12	850	30	1020	"	"	18.700	287.9	6.375	98.1		Yellowish red; clear.
13	770	27½	1017	"	"	18.480	284.5	6.160	94.8		Yellow; slight cloudy deposit.
14	640	22½	1022	"	"	20.560	316.6	5.120	78.8		" clear.
15	730	25½	1023	"	"	18.615	286.6	5.840	89.9		"
16	880	31	1021	"	"	22.880	352.3	4.840	74.5		"
17	620	21½	1027	"	"	19.220	295.9	5.890	90		"
18	550	19½	1027	"	"	17.050	262.5	5.225	80		Pale yellow; lithates.
19	620	21½	1025	"	"	15.500	238.7	4.080	62		"

Weight on the 14th day 25·2 kilogrammes or 55·5 lbs.

The average daily excretion of urinary water, urea, and chlorides during each period has been—

		Amount of water.	Urea.	Chlorides.
		C. C.	Grammes.	Grammes.
1	Before lysis (2 days' urine) .....	·1160	29·490	1·438
	or per kilogramme of body weight .....	.....	1·170	·0569
2	During lysis .....	·735	17·075	·735
	or per kilogramme of body weight .....	.....	·677	·029
3	During 1st week of convalescence .....	·671	15·898	4·920
	or per kilogramme of body weight .....	.....	·651	·193
4	During 2nd week of convalescence .....	·667	18·662	4·996
	or per kilogramme of body weight .....	.....	·740	·196

STATISTICAL DETAILS  
OF  
THREE YEARS' EXPERIENCE IN RESPECT  
TO THE  
FORM OF AMAUROSIS  
SUPPOSED TO BE  
DUE TO TOBACCO.

BY  
JONATHAN HUTCHINSON, F.R.C.S.,  
SURGEON TO THE LONDON HOSPITAL AND TO THE HOSPITAL FOR SKIN  
DISEASES; ASSISTANT SURGEON TO THE ROYAL LONDON  
OPHTHALMIC HOSPITAL, MOORFIELDS.

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Received April 29th.—Read June 25th, 1867.

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It is no part of my object in the present paper to enter into details as to the ophthalmoscopic appearances which characterise the disease known as "white atrophy of the optic nerves." Amongst ophthalmic surgeons these are well recognised and for the most part are easily distinguished from those of other diseases. In order to make the subject more intelligible to those not conversant with the ophthalmoscope, I may, however, be permitted to say a few words regarding them. The cases which form the subject of this paper are recognised by the loss of vascular supply to the optic nerve itself. There is not usually much diminution in the size of the vessels

which supply the retina, and often these remain of good size when the nerve itself is as white as paper. The first stage (one which is usually very transitory and perhaps often altogether omitted) is one of congestion, during which the disc looks too red. Then follows pallor of the outer half of the nerve disc, that part which is nearest to the yellow spot. During these stages the patient complains of dimness of vision merely. Everything seems in a fog to him, but he has no pain in the eyes nor any photophobia or photopsiæ. In a later stage the whole of the optic disc has become pale even to a blue-milk whiteness, and later still there is proof, not only of anæmia of the nerve, but of advanced atrophy. The stages usually occupy from four months to a year. In many cases the patient becomes at length absolutely blind, but in others the disease having advanced to a certain point is arrested. There is from first to last no evidence of disease of any structure in the eyeball excepting the optic nerve, and even after years of absolute blindness, the retina, choroid, &c., remain healthy and their blood-supply good. Almost always both eyes are affected and progress almost *pari passu*. Sleepiness, a little giddiness, and a little headache are usually the only constitutional symptoms which attend it, and these disappear at a later stage and the patient regains his usual health. As there is no tendency to fatal complications, opportunities for post-mortem examination of the brain are scarcely ever obtained.

White atrophy of the optic nerve is observed in another class of cases which are quite distinct from those which are the subject of this paper, but which under certain conditions are distinguished from them with difficulty. I allude to those in which the atrophy is secondary to acute inflammation of the nerve-structure. During the stage of inflammation these cases are easy enough of diagnosis, the evidence of lymph on the disc being positive and readily appreciated. At a later period, however, when the lymph has been absorbed, the appearance of atrophy may closely simulate that presented by the cases in which atrophy is the primary change.

There are still some points of difference in the state of

the disc which will help the trained observer, and the history of the early symptoms is usually very different. In optic neuritis we usually have severe headache, repeated attacks of sickness, implication of other cerebral nerves, or even epilepsy or hemiplegia. The cases of primary atrophy are on the contrary characterised by the absence of all other serious symptoms.

In submitting any question to the test of numerical inquiry, everything depends on the accuracy of the materials collected. In the present instance the chief risk of fallacy concerns cases of atrophy after neuritis. If these cases should, in error, be counted with those of primary atrophy, very different statistical conclusions would be arrived at from those which I have obtained. Neuritis is common in children and women, whereas primary atrophy is, I believe, almost unknown in children, and very rare in the female sex.

With an apology for these introductory statements I will now proceed to the subject-matter of my paper.

Three years ago I collected all the cases of uncomplicated cerebral amaurosis of which I had the notes, with the object of determining, as far as such statistics could help me, the supposed influence of tobacco in the production of this disease. The most remarkable fact which my cases showed was the infrequency of the disease in women. Excluding all cases in which the amaurosis was clearly secondary to optic neuritis, those in which there was disease in the eyeball itself, and those in which only one eye was affected, I had to deal with a total of forty cases. In all in this group the disease was symmetrical, of gradual progress, without any other marked symptoms of cerebral disease, and tending slowly to the extinction of sight with white atrophy of the optic nerves. Of these, thirty-seven were in men and three in women. All the patients which I here include were adults, and almost all the men had been smokers. This fact as to the very different proportions in the two sexes impressed me strongly as of great value as a possible clue to the cause of this obscure malady, and I ventured to discuss at some length those differences in the habits and conditions of the two sexes



which might be supposed to have possibly acted as such. This inquiry did not, however, yield much result, or rather its results were negative. In a large majority of my patients it was not possible to assign any definite sexual cause for the disease. In but few was there any reason to suspect excessive sexual indulgence, the existence of spermatorrhœa, or the injurious use of stimulants. The occupations of my patients had been extremely varied, so much so as to entirely baffle conjecture in this direction.

This former report was compiled under several disadvantages. In some of the cases the diagnosis had not been made as accurate as could have been wished, and, as many had been under care before I was aware of the importance of the tobacco question, I had omitted inquiries on this head.

During the three years which have since passed I have continued, with the able assistance of my friend Mr. Waren Tay, to carefully record all facts bearing upon the subject which came under my notice. To these I now invite the attention of the Society. In the appended tables will be found a brief statement of the leading facts as to all the cases of symmetrical amaurosis without neuritis which have been under my care between January 1864 and December 1866. I have excluded all cases in which any evidence of inflammation of the retina, choroid, or optic nerve was to be found, and also all cases in which but one eye was affected. These latter belong clearly to a different class.

My present report comprises thirty-seven cases. In all, the patients were adults, and at ages varying from 21 to 56, and averaging 40. Almost all the patients were in good health, and in but few had there occurred at any stage any symptoms of cerebral disorder apart from the amaurosis. None of them were paralysed in any way, and none of them presented any material defect in hearing. In most there was the history of some frontal headache, slight giddiness, &c., at the time the first defect of sight was noticed, but in none had these symptoms been severe, and in almost all they had ceased as the amaurosis advanced. Of the thirty-seven cases, thirty-four occurred in men and three in women; thus remarkably

confirming the conclusion on this point arrived at in my former paper. Here I may perhaps be allowed to state that I have been scrupulously careful to include all cases occurring in women which could be fairly considered of the same class as those in men. Most of my cases have been observed in public, many of them have been made the subjects of clinical demonstration at our evening lectures at Moorfields, and I have taken every opportunity of directing attention to the exceptional cases in which females were the patients.

I have arranged the cases in two tables, one of females the other of males, and will append a few comments on each.

Most of the abbreviations used in the table will, I trust, be found intelligible. It is, however, necessary to explain that whenever the statement "vessels of good size," or the like, occur, only the branches of arteria or vena centralis are referred to.

TABLE I (*Females*).—*Cerebral amaurosis in women (three cases)*.

Number, reference, and date.	Name, occupation, state of health, &c.	Age, duration of amaurosis.	Use of tobacco, alcohol, &c.	State of eyes.	Other symptoms.	Progress and remarks.
1 March, 1865	Eliza N—, married 11 years, good health, menstruation regular	29; 4 months	Temperate. No cause assignable	Could only read letters of No. 19. Discs pale	Gradual failure. Bad headaches, muscæ and photopsiæ occasionally, often giddy	This case very closely resembles the cases we meet with in men. She attended six months, and got slowly worse until she could only count fingers.
2	Mary Anne H—, a widow 16 years, a laundress, menstruation ceased five years, thin and rather feeble	52; 2 years	Had used snuff, but denied that she had smoked. No cause assignable	Quite blind. Discs blue white. Central vessels of fair size	Gradual failure during two years; a little headache, but no material head symptoms	A very close parallel to what we meet with in men.
3	Mrs. C—, looking healthy	27; 3 months	Had suffered severely from constitutional syphilis seven years before the amaurosis began. No use of tobacco or abuse of alcohol.	Could only read 70 at 1'. Discs almost normal, a little paler than natural. Pupils of medium size, and fairly active	Had suffered for six months from head-ache. For three months her sight had been failing. No sickness or other symptoms	I think there can be but little doubt that this is a case of syphilitic neuritis in which the lowest parts of the nerves are not as yet involved.

Two of the cases in which women were the subjects, Nos. 1 and 2, present close parallels to what we meet with in men. In one, the patient was a married woman of 29, in the other a widow of 52. In neither could any special cause be assigned. Both denied having ever smoked, but one of them, the older one, had for years been accustomed to use snuff. In one, the patient came under care when quite blind, with the nerves in a state of advanced atrophy, in the other, I had the woman under care for a year, the disease steadily advancing in spite of treatment. In the third case the patient was undoubtedly the subject of constitutional syphilis and it may be suspected that the failure of sight was in connection with that cause. When she was under observation, six months from the commencement of her symptoms, her discs showed but little deviation from the normal state although she had already so far lost her sight as to be only able to read the largest capitals, (Sneller, 70 at 1'). Under treatment, by iodide of potassium, some improvement is, I think, in progress. This case is especially important as illustrating one of the possible causes of cerebral amaurosis without neuritis, or rather without proof of neuritis on the optic disc.

TABLE II (*Males*).—*Cerebral amaurosis in men (Thirty-four Cases)*.

A statement of the details of thirty-four cases in which men became the subjects of uncomplicated amaurosis from white atrophy, being all that came under my care during a period of three years (January, 1864, to December 31st, 1866).

Number, reference, and date.	Name, occupation, and state of health.	Age, and duration of amaurosis.	Use of tobacco and alcohol.	State of eyes.	Other symptoms.	Progress and remarks.
1; Nov. 5, 1866; C. 111	John J—, shoemaker, married; in good health	37; 5 months	Half an ounce of hashag daily. Very temperate in the use of stimulants	200 (Sn.) at 20'. Discs very white. Central vessels much diminished	In good health all the time.	
2; Nov. 1, 1866; C. 95	James E—, a sawyer, married; florid, but nervous	46; 3 months	Half an ounce daily for twenty-seven years. A spirit drinker	Pupils sluggish, of normal size. Reads 18 (J.). Outer parts of discs grey. Retinal veins turgid	A florid, robust man; tremulous and nervous from drink. Believe they had nothing to do with the amaurosis.	There were curious crucicents in each eye, but I believe they had nothing to do with the amaurosis.
3; Oct. 29, 1866; C. 92	George H—, short, spare; healthy	41; 2 months	A quarter of an ounce daily. Temperate in stimulants	Pupils normal. Just spells out 16 (J.). Discs scarcely paler than natural	No other symptoms.	
4; Oct. 15, 1866; C. 82	William Y—, a labourer; healthy	47; 2½ years	Half an ounce a day for twenty years. Temperate as regards stimulants	Pupils rather larger than natural, and almost motionless. Discs grey. Central vessels somewhat diminished. Quite blind	Had some pain in the temples when the symptoms began. In nine weeks was quite blind	He left off smoking six months after the amaurosis began; he thought it had made him nervous. This man fancies that after two years' blindness he is now beginning to see shadows.

5; Aug. 20, 1866; C. 66	Benjamin P—, stunted growth, pale complexion, aspect of defective virility	20; 5 years	Very little for two years	Puzzles out large capitals with left. Right quite blind. Dises white, more so at outer parts. Ves- sels almost of natural size	No other symptoms	He doubts whether he ever could see with the right eye. He never smoked till after his eyes began to fail. Six months later he could scarcely see to count fingers; pupils small and fixed.
6; July 6, 1866; C. 42	Robert Y—; heal- thy	55; 4 months	Smoked for twenty years. Half an ounce a day for three or four years. Three or four pints of beer a day	Puzzled out the largest letters (J.). Extreme pallor of outer part of each disc. Vessels of nor- mal size	Had great diffi- culty in learning to smoke, and gave it up several times be- cause it made him ill	
7; May 31, 1866; C. 20	James H—, nurse, Haalar Hospital; healthy; married	40; 2 years	Was accustomed to smoke—quantity not light. Had been out side smoking more just of before eyes failed	Rare perception of light. Grey atrophy of outer side. Vessels clearly defined	Chancres and rash eighteen years ago. No relapse. Only three out of ten chil- dren have lived	He said he had often been made ill by smoking and had left off on that account, but began again. One cigar would make him ill for days.
8; Nov. 19, 1866; C. 15	Thomas J—, shoe- maker, soldier; mar- ried; healthy	30; 6 weeks	A quarter of an ounce daily for eight years	Pupils large, equal, and act fairly. 100 (Sn.) 20'. Congestion; more especially of left	No syphilis. No other symptoms.	
9; April 30, 1866; C. 1	George W—, shoe- maker; fair health; widower for 11 years	36; 4 years	Had never smoked	Pupils small, equal, very sluggish. Largest letters (J.). White atrophy. Veins large; arteries much dimi- nished. Whitest next yellow spot	Liability to epis- taxis (hereditary) and giddiness	Had been troubled with muscae and "rainbows." His epistaxis had often occurred several times in the week. The amaurosis had advanced very slowly.

Number, reference, and date.	Name, occupation, and state of health.	Age, and duration of amaurosis.	Use of tobacco and alcohol.	State of eyes.	Other symptoms.	Progress and remarks.
10; April, 1866; B. 390	J. S—, a Jew merchant, from Ha- vannah; good gene- ral health	56; 1½ year	Heavy drinker and smoker	Well-marked white atrophy. So far ad- vanced that he could not direct his eyes as required.	None	January, 1866.—Has left off tobacco and taken iron and strychnia. No. 11 (J.). With + glass reads No. 6 (J.). Without glass 40 (S.) 20'. Considers himself much better since he left off smoking.
11; Oct. 27, 1865; B. 366	James P—,	42; 5 months	Half an ounce daily	Both larger than natural; fairly active. No. 19 (J.). Discs paler next yellow spot	None	
12; Sept. 25, 1865; B. 353	Francis S—; un- married; healthy; from Jersey	34; 2 years	Smoked nearly all day long for eighteen years	Pupils widely dila- ted and fixed. Lar- gest letters at 20'. Pallor of disc. Whiteness next yel- low spot	No other symp- toms. Gradual fail- ure.	
13; Oct. 9, 1865; B. 341	Thomas L—, la- bourer; hands tremu- lous	25; 11 months	An ounce daily for four years	Letter or two of 20 (J.). Artery of tolerable size. Vein not turgid. Centre depressed, dirty grey. white; edges not dis- tinct	Severe headaches for three to four months. Chancres just before eyes failed. No secondaries. Blow on his head, which stunned him, a fort- night before his sight failed.	

14; June 8, 1865; B. 294	George W—	31	Half an ounce daily for three or four at years distance. No. 10 (J.) prox.	Only largest letters		
15; May 29, 1865; B. 284	Henry K—, mar- ried; gardener	50; left 6 months, right 6 weeks	A quarter to half an ounce daily for thirty years	Moderately dilated, sluggish; left, just see light; right 20 (J.). Discs very white; margins ab- rupt; vessels dimin- ished. Left much whiter than right		
16; Jan. 28, 1865; B. 241	James R—, single, clerk; very healthy	30; 5 months	Smoked and chewed half an ounce a day for the last fifteen years; tance. 16 (J.) prox. ounce daily last five years	Cannot make out largest letters in dis- tance. 16 (J.) prox.	—	Left off smoking Dec. 22, 1864. Nov. 30, 1865, largest letters at 20 feet. 6 (J.) at 5½".
17; Nov. 4, 1864; B. 235	Thomas K—, watchmaker, mar- ried; pale, not speci- ally ailing	50; 10 months	Half an ounce daily; a great drinker	Large, exceedingly sluggish. Largest letters at 20; grey- ish-white, margins abrupt and irregular; vessels half natural size	Steady failure after an attack of delirium and atrophy of nerve, while he supply to retina remained his blind-good. May 11, 1865.—Left of pupil twice the size of the right, which is larger than numbness and weak- ness of both lower extremities. Both 14 (J.).—Complained of testes atrophied and numbness of hands and feet. flabby. Abeyance of sexual appetite for some months	A marked case of anemia and atrophy of nerve, while supply to retina remained good. May 11, 1865.—Left of pupil twice the size of the right, which is larger than numbness and weak- ness of both lower extremities. Both 14 (J.).—Complained of testes atrophied and numbness of hands and feet. flabby. Abeyance of sexual appetite for some months Loss of smell.



Number, reference, and date.	Name, occupation, and state of health.	Age, and duration of amaurosis.	Use of tobacco and alcohol.	State of eyes.	Other symptoms.	Progress and remarks.
18; Dec. 29, 1864; B. 239	George C—, shipwright; married	49; 7 weeks	Regular smoker; quantity not stated. Temperate in beer, &c.	Left dilated and fixed, slight divergence; sees only tal shadows; discs blue-white and depressed, margins distinct, vessels diminished. Right less advanced than left, and he can see capitals with it	Various blows on head; attacks of frontal headache and dim-vision. Blow below left eye from copper nail eight weeks ago	Continued at work till week before admission. March 9, 1865.—In good health; cannot count fingers; not smoked since last visit: hands and feet numb (pins and needles). Discs abruptly margined and white, artery still easily seen. May 1.—Numbness continued. May 14, 1866.—Excellent health. No perception of light for nearly 12 months; no pain. Weight and numbness about abdomen; could pick up a pin. Ophthalmic examination.—Same as before.
19; Oct. 20, 1864; B. 280	William R—, publisher	45; Right 12 months; left 6 months	Half an ounce daily	Both discs white	Violent earache 12 months ago, with giddiness and overpowering sleepiness. No headache	Continued attending till February, taking strychnia. Dec. 11, 1866.—Could scarcely tell light from darkness; had gained flesh; good health; could walk twenty miles a day; left off smoking; pupils of equal size and decidedly small, absolutely motionless. With atropine

20; Jan., 1866; B. 221	John H—	47; 4 years	Freemoker; quantity not stated	White atrophy of both optic discs	dilated well. Discs abruptly margined, dirty-grey. Vessels dilated, almost of natural size. Could direct his eyes as desired.
21; June 9, 1864; B. 203	Thomas C—, farmer; weak and pale	46	Free drinker and smoker all his life	Both discs white; quite blind	Gradual failure. Myopic. Dizzy. Eight years ago a fit with failed several years before hemiplegia. He quite recovered. Sexual functions freely indulged, but now in abeyance. No history of syphilis. Variocoele
22; June, 1864; B. 200	William McC—, boatman; married 8 months	21; Sight failing 8 months; quite blind 6 months	Smoked and drank freely, but left off both before the eyes failed	Pupils dilated and fixed; optic discs white, abrupt; artery very small, vein much diminished	According to his statement the failure of sight did not commence until four months after he had become a total abstainer, and also left off smoking. It progressed very rapidly, and he had some unusual symptoms.

Number, reference, and date.	Name, occupation, and state of health.	Age, and duration of amaurosis.	Use of tobacco and alcohol.	State of eyes.	Other symptoms.	Progress and remarks.
23; Mar. 24, 1864; B. 186	J. R. P—, clerk; pale, rather feeble	51; Sight failing 12 months	Pipe night and morning; very steady man	Grey-white, espe- cially towards yellow spot; vessels of mode- rate size	Gradual failure; giddiness occasional- ly. No other symp- toms	Dec., 1865. —Could not count the windows, yet was annoyed by the gaslight and sunlight. Pupils equal, natural size, quite motion- less. Good general health. Ophthalmic examination as before.
24; April 14, 1864; B. 184	J. J. G—, sailor; healthy	20; Sight failing 8 months	Half an ounce daily for three years	Normal, act fairly 20 (J.). Discs grey- white; arteries very small	Simple failure. No other symptoms	Great difficulty in learn- ing to smoke, and also suf- fered much from sea-sick- ness. April 2, 1866. —Vision as before; health good. "Aw- ful headaches" for two days once in three weeks. Left off smoking after first visit, but has taken to it again.
25; Mar. 24, 1864; B. 176	Henry G—, pilot on Thames; stout, healthy looking; mar- ried 8 years	31; Sight failing 3 months	Smoked an ounce or an ounce and a half a week for 13 years; very steady	Discs abrupt, grey- white; vessels fair dimensions	Gradual failure. No other symptoms.	Great difficulty in learn- ing to smoke. Liable to sickness after smoking more than usual. April 28. — Much the same.
26; B. 15	John B—	56; Sight failing 6 months	Three quarters of an ounce daily	White atrophy	Gradual failure. No other symptoms.	

27; Dec. 11, 1865; B. 134	John M—	53; Sight failing 15 months	Not stated	Atrophy commencing next yellow spot; artery much dimin- ished	Gradual failure	The notes of this case are defective.
28; Mar. 8, 1866	William W—	37; Sight failing 1 month	An ounce a day for 5 or 6 years	Could still puzzle out letters of 2½ (Snellen)	Gradual failure	
29; June 12, 1866; B. 275	Isaac M—, quite healthy; married	35; Sight failing 12 months; nearly blind	Two ounces a week since st. 19; tempe- rate	Pupils medium, very sluggish; white atrophy, abrupt; ves- sels of moderate size. Pyramidal cataract right only. Could just see a hand before him	Gradual failure; no varicocoele. No other symptoms.	Continued to smoke. His report (by letter) in August 22 was that he got worse.
30; Oct. 29, 1863	H—, a farm-la- bourer; married 3 years; good health	26; Sight failing 10 months	An ounce a week for ten years; steady and temperate in other indulgences	Could not see the largest letters. Both discs white	No other symptoms of cerebral disease. Gradual failure of sight. Small double varicocoele, but no loss of sexual vigour	
31; Dec. 20, 1864	Philip B—, stout, florid, very strong, and in excellent health	33; Sight failing 16 months	Half an ounce a day for 15 years; very temperate	Could not see the largest letters. Pupils moderately dilated and very sluggish; discs blue-white and well defined. Central vessels of good size	Slight giddiness and occasional head- ache at the time the eyes began to fail	He remained under treat- ment for a year without material change. He attri- buted his disease to his having changed his occu- pation and been less in the open air.

Number, reference, and date.	Name, occupation, and state of health.	Age, and duration of amaurosis.	Use of tobacco and alcohol.	State of eyes.	Other symptoms.	Progress and remarks.
32	Patrick B—, an Irish carman; in excellent health; now living at Selby, Yorkshire	50; Sight failing for 2 years	Half an ounce a day for 20 years, and free allowance of whisky	Total blindness for three months. Both eyes pupils sluggish; left normal size, right curved twice as large. Both discs quite white and atrophied. Central vessels of fair size	Sleepiness, giddiness and constant ptyalism occurring in the beginning of the failure of vision. He had formerly been a currier in Ireland and constantly in the open air. A year before his eyes failed he gave up this and came to a more confined occupation in England.	The ptyalism which occurred was a very interesting symptom. It continued up to the time I saw him, and annoyed him very much. He had formerly been a currier in Ireland and constantly in the open air. A year before his eyes failed he gave up this and came to a more confined occupation in England.
33; Nov. 29, 1866; C. 119	Thomas W—, in good health	38; Sight failing for some months	Half an ounce a day for twenty years. Free allowance of beer	Could not see the largest letters. Right disc pale; left grey-white and indistinct from slight neuritis (?)	Pain in forehead; gradual failure of sight	One disc showed doubtful evidence of neuritis, but there had been none of the usual symptoms of that disease, and there was no inflammation in the other eye.
34; Nov. 29, 1866; C. 120	James H—, cabman; in good health	38; Sight failing 12 months	Half an ounce daily for many years. Has drunk spirits and ale —chiefly ale	Right could read 8 lines with +16. Hypermetropia. Pupils sluggish; left disc very pale; right somewhat pale	Gradual failure of cerebral symptoms	In this instance we saw the patient in an early stage of the anaemia of the discs.

I may conveniently classify the cases in men into several groups, observing first that *all* the patients were men in the prime of life, and that their pursuits were so various that no advantage could be obtained from stating them here. In no single case, however, was the patient engaged in working in tobacco.

GROUP I.—*Men who had smoked, and in whose cases no other special cause was assignable.*—This includes twenty-seven<sup>1</sup> cases out of the thirty-four. All these patients had been heavy smokers, consuming, many of them, from half an ounce to an ounce a day. Most of them had, as far as I could ascertain, been moderate in the use of alcoholic stimulants. Four had been free drinkers. In all these cases the course of the disease had been much the same; progressive towards white atrophy. In none, as far as I am aware, has any other form of nervous malady as yet shown itself.

GROUP II.—*Men who had smoked, but in whom other causes were suggested.*—Five cases come into this group, and each must be briefly mentioned.

In Case 7, the patient had suffered from constitutional syphilis, but it was eighteen years ago, and he had no tertiary symptoms. It seemed not improbable that tobacco had been the cause, for he had persisted in smoking, although it had frequently disagreed with his health.

In Case 13, the man had had a chancre just before his sight failed, but no secondary symptoms, he had also had a blow on the head. It is scarcely probable that either of these events was the cause of the amaurosis.

In Case 17, an attack of delirium tremens preceded the amaurosis, and entire failure of the sexual function with a degree of loco-motor ataxy attended it. This man had smoked largely.

<sup>1</sup> The cases numbered in the table, 1, 3, 4, 8, 11, 12, 14, 15, 16, 19, 20, 23, 24, 25, 26, 27, 28, 29, 30, 31, 32, 33, 34, are those in which the patients had been heavy smokers but not intemperate in drinking. Cases 2, 6, 10, are also counted in Group I, but in them the patients had been intemperate in drinking as well.



In Case 18, the man believed that a blow on one eye had been the cause of his blindness, but there was little or no evidence in support of this conjecture. Both eyes suffered, and he had smoked freely.

In Case 21, the disease had progressed irregularly, having affected one eye three years before the other suffered. The patient, a heavy smoker and free drinker, had suffered from transitory hemiplegia eight years before. He had been addicted to very liberal sexual indulgence, and had entirely lost his aptitude in this matter during the progress of his amaurosis.

In two of the five cases it is quite open to conjecture that tobacco was the cause of the complication as well as of the amaurosis, and in the other three the possible cause mentioned was by no means a probable one.

GROUP III.—*Men in whom it seems scarcely possible that tobacco could have been the cause.*

In Case 5, the patient was aged 20, but looked like a boy of 14; his virility was, I think, certainly in abeyance. He had smoked a little for two years, but he assured me that his eyes had been failing several years previously.

In Case 9, a shoemaker, a widower, who had never smoked, had suffered from slowly progressive amaurosis for four years, but was still able to read large type. This man had been liable (hereditarily) to frequent attacks of profuse epistaxis, and during the progress of the amaurosis had been troubled with muscæ and rainbow halos. In its slow progress, and the phenomena just mentioned, his attack had differed from the usual type.

Case 22 is very difficult to deal with. The patient, a boatman from Yorkshire, had been accustomed to smoke very freely indeed, and also to drink freely. He asserted, however, that four months before his eyes began to fail he had become a total abstainer, and also left off smoking. The chief failure occurred during the two months after his marriage, and was such that he was obliged at once to give up his employment; but he asserted that his eyes were decidedly



failing ten days before his marriage, and he did not believe that that event had had anything to do with it.

On other points I must leave the cases as detailed in the table to explain themselves. As regards the influence of *abstinence from tobacco* (as a measure of treatment) upon the progress of this form of amaurosis, although I have no very definite statements to make, I have formed strong impressions. During the last three years I have held it to be a bounden duty to warn all who present the symptoms of this disease against smoking, and in only a few instances (provided the patient was seen early) did the disease afterwards progress to blindness. The impression which I had formed, before my attention was directed to tobacco as a cause, was that this disease invariably, in spite of treatment, went on to complete blindness.

The importance of the subject may be in some degree estimated by the frequency of the disease. Amongst my patients at Moorfields an average of one new case of this form of amaurosis is presented every month. My practice probably represents about a ninth of that of the Institution, and it is therefore fair to assume that not fewer than a hundred cases come before its staff every year. It is to be recollected that almost all of them are male adults in the prime of life, most of them the heads of families. I have no wish to express prematurely an opinion as to whether tobacco is, or is not, the real cause. The facts must be left to have their due weight. To find that in upwards of eighty cases we have a disproportion in the two sexes of one to eight is certainly somewhat startling, and the circumstance that in the great majority no other assignable cause can be discovered does not tend to remove suspicion.

Some of the cases support the belief that where tobacco acts injuriously there is a special idiosyncrasy as regards it, inasmuch as the patients stated that they had experienced unusual difficulties in learning to smoke.



A C A S E  
OF  
ANEURISM OF THE FEMORAL ARTERY,  
THE  
SAC OF WHICH BURST, AND ITS TREATMENT;  
WITH OBSERVATIONS.

BY  
JOHN BIRKETT, F.R.C.S.,  
SURGEON TO, AND LECTURER ON SURGERY AT, GUY'S HOSPITAL.

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ON the 7th of April, 1867, a man, *æ*t. 40, was admitted into Guy's Hospital under my care, on account of a large and very painful swelling, occupying the whole of the front of the right thigh. Pulsation, synchronous with the action of the heart, was distinctly felt in almost every part of it; as well as the peculiar increase of size at each contraction of the left ventricle which usually accompanies a tumour in immediate communication with the interior of a large arterial trunk.

The man himself gave an account of the progress of the disease, for he had never before this time sought surgical assistance. For about eight months he had felt a swelling in front of the right thigh, which beat "like your pulse, you know," these were his words. It increased slowly; had been almost painless, and had never obliged him to desist from his

usual labour. Whilst at work, during the forenoon of the day of his admission, he felt something snap in his thigh, at the site of the long noticed swelling, and almost immediately afterwards the limb became swollen and very painful.

He was unable to continue his occupation, and maintained a state of repose, but rubbed the swelled part with a liniment. The pain and swelling increased, and he entered Guy's Hospital about ten hours after the "bursting of the swelling," as he styled it.

I saw the man about 8 o'clock in the evening, a short time after admission. The integuments of the right thigh were tense, almost as if ready to burst; by firm pressure the tumour felt elastic, and pulsating movements of the contents were very distinct. There was not a trace of pulsation to be felt in the popliteal artery or its branches. The femoral pulsated strongly above the swelling. The foot and about half the leg were œdematous.

There was not the slightest difficulty in discriminating the character of the disease: it was too palpable to admit of doubt.

As regards treatment, three methods suggested themselves:

1. Amputation of the limb above the aneurismal sac.
2. Deligation of the external iliac artery.
3. To incise the sac of the aneurism and apply a ligature on the vessel entering it from above, and another on that leaving it below.

The last operation I performed as follows: I made an incision into the sac about six inches long, the one through the integuments being about ten inches. Its whole course was parallel with the fibres of the sartorius muscle. The contents of the sac escaped at once, so that the openings of the artery above and below it were immediately seen. The hæmorrhage, however, was completely arrested by Mr. Cooper Forster, who had charge of a tourniquet applied over the abdominal aorta, and by the pressure he made upon the external iliac artery. A flexible catheter was passed into the lower division of the femoral artery; a ligature was quickly

placed around it and tied after the catheter was withdrawn. Without disturbing the relations of the femoral artery above the sac more than was absolutely necessary, I passed a ligature beneath it with great facility, and tied it about half an inch above where the sac commenced.

The apertures of the arterial trunk were about three inches apart, and a layer of arterial tissue connected them and formed the posterior wall of the aneurismal sac. Upon this, and especially towards the upper orifice, there was earthy, atheromatous deposit.

Very little fibrine was seen in the sac.

Sutures were used to bring together the edges of the wound in the integuments at the upper and lower ends; the centre was allowed to gape, although the whole was supported by strips of moist bandage. The limb was enveloped in a flannel bandage, and a pillow was placed beneath the popliteal region, so that the member might repose in the most easy position.

During the following twelve hours the chloroform produced a nauseating effect, and he vomited slightly.

On the 8th there was no constitutional disturbance; there was a slight escape of serum from the wound, and the temperature of the foot and leg was normal.

9th, second day.—He slept well during the night, and his condition was undisturbed. The wound of the parts not infiltrated with blood was quite healthy, and beginning to heal. Removed two sutures, those nearest the centre of the wound, which allowed a more free escape of serum. Temperature of the limb normal.

10th, third day.—Very well. Feels hungry, and ate a small mutton cutlet. The ends of the wound healing. Removed the two remaining sutures. The centre of the wound, the site of the sac, and part of the sartorius muscle, which was infiltrated with blood, were sloughing. To discontinue the wet lint, and to use strips of bandage bathed in a solution of permanganate of potash, and to syringe the wound frequently with that solution. This local treatment was assiduously carried out by the dresser of the case, Mr. William Toulmin,

and the progress to convalescence was uninterrupted for several days. Generous diet and stimulants were liberally allowed.

16th.—On this day, the ninth from the operation, a large slough came away. The wound was healing rapidly, and the man's general health scarcely affected.

17th.—This being the tenth day, the lower ligature came away, and from this date there was nothing deserving of remark until the

22nd—or the fifteenth day after the operation, when the upper ligature separated without bleeding even from the granulations by which it was surrounded. The surface of the whole wound was covered with the healthiest of granulations, from which there was very little suppuration, and the ends of the wound had cicatrized. The last day or two he had been somewhat troubled with a cough, and as he did not restrain the action of his abdominal muscles in that effort, but, on the contrary, used much force, I cautioned him, and directed that he should place digital pressure over the region of the femoral artery, below Poupart's ligament, when the fit of coughing was violent. This he did frequently, but on the

24th—about 4 a.m., whilst coughing, he felt blood trickling down his thigh from the upper end of the wound. This was the seventeenth day from the operation, and the second since the upper ligature came away.

The hæmorrhage was easily controlled by pressure, but before its application he had lost much blood. When I saw him, about ten hours afterwards, his face was blanched, his pulse weak, and he was very desponding. I told him I must again stop the bleeding by tying the artery, to which, after exhibiting slight reluctance, he consented. Chloroform was administered, and by merely breaking down the recent adhesions with the finger, the open mouth of the vessel from which the blood flowed was soon discovered. Pressure was, of course, made upon the external iliac artery. The trunk of the vessel was rather firmly united to the surrounding tissues, and its separation from them was effected with some difficulty. A ligature was passed around the vessel and tied.

This checked all bleeding, but the vessel did not pulsate at the site of the ligature, nor for half an inch above it, although above that point the beat of the artery was distinctly perceptible.

25th.—Passed a quiet night, although he was rather nauseated by the chloroform. He had quite recovered his usual spirits, and spoke hopefully of recovery.

30th.—Progressing favorably, although his appetite is indifferent. The stimulants have been continued. The wound is again closing up, and the suppuration moderate and healthy.

May 2nd.—Last evening there was a trifling escape of blood from the upper end of the wound, and the dresser found the ligature detached. This was the eighth day from its application. When I saw the man, at 2 p.m., there had been no more hæmorrhage; the wound was healing, and its appearance was quite healthy. The whole limb was bandaged, and a compress applied over the femoral artery.

5th.—Cicatrization advancing rapidly; appetite improved. To sit up in bed, supported by pillows. The bandage discontinued.

9th.—At 1.30 a.m. there was bleeding to about two ounces from the upper end of the wound, which ceased before the house-surgeon reached him. A graduated compress was, however, applied over the artery as before.

13th.—There has been no bleeding for four days; he eats well, taking meat. The wound is nearly healed.

During the subsequent weeks of May he was troubled with rheumatic pains in the arms and trunk; the wound, however, healed favorably, and in the last week he was able to leave the bed and walk about the ward. In fact, the wound was healed in eight weeks.

He was discharged from the hospital on the 3rd of June, fifty-seven days after admission.

*Remarks.*—This patient had pursued the occupation of a sand-dredger on the Thames for a period of twenty years. It is one attended with unusual exposure to the vicissitudes of

weather, and requires great bodily exertion. He had always lived very freely, partaking daily of abundance of animal food, as well as stimulants. The general nutrition of the man was very good.

*Treatment.*—At the commencement of the narration of the case I briefly alluded to three plans of treatment.

1. Amputation of the limb. This operation I considered to be pregnant with risk, and almost certain to be followed by a fatal termination, for the following reasons. The incisions must have been made close to the hip-joint, and surgeons generally admit that primary amputations near to the pelvis are rarely attended with success. Besides, it seemed to be the duty of the surgeon to take other measures, and to attempt to save the limb before resorting to that expedient; in fact, to keep that procedure in reserve.

2. Deligation of the external iliac artery could have been easily performed. It would have been a comparatively bloodless operation. But the following objections I regarded to be paramount. I might have arrested a large supply of blood to the limb, and, on the other hand, the flow of blood to the aneurismal sac might not have been impeded. For, as the disease had existed some months, it was reasonable to assume that the collateral branches about the pelvis were probably dilated, and by these channels the stream of blood would be conveyed to the member. From the great local tension there was reason to fear extensive sloughing in the neighbourhood of the aneurism, as well as of the tissues below it, which were even already congested and œdematous, thus affording sufficient evidence of venous compression.

Therefore, acting on the principle that the aim of the surgeon should be the eradication of the local disease, I decided to perform the operation above described.

3. In thus having recourse to the oldest method adopted for the treatment of aneurism, I was not unmindful of the difficulties I might have to encounter, and which are so fully described in all monographs upon this subject. But since the introduction of chloroform the mental calm and repose



which the surgeon experiences when he knows that the patient is not suffering at his hands, enables him to meet and overcome difficulties which were appalling to our predecessors, and to adopt measures, especially in a case of this kind, almost impracticable before. I allude to pressure with a tourniquet on the abdominal aorta. By this instrument hæmorrhage was entirely arrested.

The operation itself was completed with the utmost facility. As soon as the blood and coagula were removed, the open mouths of the femoral artery at the upper and lower end of the sac were immediately seen. I preferred the introduction of the flexible catheter within the artery in place of a probe, because it slightly dilates the vessel, and at the same time fixes it. When thus the artery is rendered steady by an assistant holding the catheter, the ligature is guided round the vessel with great ease. If an ordinary probe be used, the artery rolls about upon it, and it is not so easily separated from the surrounding connective tissue.

In the treatment of the secondary hæmorrhage, which happened two days after the separation of the upper ligature, I acted upon the principle propounded by Mr. Guthrie, namely, that of securing the bleeding vessel from the original wound, instead of tying the trunk at a distance and inflicting a second one. In doing this no real difficulty was found.

4. Immediately after the operation, when the man had sufficiently recovered from the nauseating effects of the chloroform, he was allowed to select his diet, and to take as much food of a nourishing kind as he wished. Stimulants were allowed liberally, and a favorite beverage seems to have been rum diluted with milk. Rum he preferred to brandy, therefore he had it. As soon as he wished for porter it was given him, and he consumed about two pints per diem on the average. At one time his appetite for animal food failed entirely. He then made choice of stewed eels, which was the only food he had for a few days.

Ferruginous tonics were administered very soon after the operation.

11

12

13



# of Guinea-pigs with Expectore

WEIGHT

No.	VI.	VII.	VIII.	
Material inoculated.	Phthisis expectorations. Jane N—, chronic third stage. Expectorated Feb. 15.	Phthisis expectorations. John S—, advanced second stage. Expectorated Feb. 15.	Phthisis expectorations. M. C—, commencing chronic second stage. Expectorated Jan. 15.	Phthisis expectorations. M. C—, commencing chronic second stage. Expectorated Jan. 15.
	Feb. 12, 1867	Feb. 16, 1867	Jan. 17, 1867.	Feb. 17, 1867.

ON THE

INOCULATION OF ANIMALS

AS A

MEANS OF DIAGNOSIS IN TUBERCULAR PHTHISIS.

BY

WILLIAM MARCET, M.D., F.R.S.,  
FORMERLY ASSISTANT-PHYSICIAN TO THE WESTMINSTER HOSPITAL.

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WE are indebted to Dr. Villemain for the very remarkable discovery that tubercular phthisis can be inoculated from man to animals; rabbits and guinea-pigs, which are naturally liable to the disease, being fit subjects for inoculation.<sup>1</sup> This gentleman inoculated twenty-two rabbits with tubercular matter; only two escaped the disease, and in one of these death appeared to result from the method of the inoculation—the tubercular matter having been injected into the trachea. He also inoculated two guinea-pigs with human tubercular matter; on the examination of their bodies, one two months and the other three months after inoculation, they were found to exhibit tubercles. This gentleman observes that two rabbits which lived with those inoculated, but left sound, on being killed and examined ex-

<sup>1</sup> 'Gazette Hebdomadaire' for December, 1865, and November and December, 1866.

hibited no tubercles; on another occasion, a rabbit which had been kept throughout with inoculated animals had both sciatic nerves cut, which was followed on the 24th July with a chronic suppuration and swelling of the tibio-tarsal articulation, caries of the calcaneum, and extreme emaciation. This animal was killed on the 21st of November, when it was found to exhibit no signs of tubercles. Again, he inoculated a rabbit with the intestinal contents in cholera and pus from an abscess, which failed to cause the formation of tubercles.

Dr. Villemain concludes from his experiments that tuberculosis is a specific disease, and so far allied to syphilis and glanders.

As to the incubation of the disease, he states that ten days after inoculation, a rabbit exhibited one granulation in the lung; in the lungs of another, only two tubercles could be detected twenty days after inoculation; a third, after being inoculated for twenty-eight days, was found to have a considerable number of tubercles in its lungs, spleen, and kidneys. He also observes that the young of inoculated rabbits are occasionally still-born, and those brought forth alive die apparently of starvation, from their mothers having no milk for them.<sup>1</sup>

Mr. Simon, who followed up Villemain's experiments and communicated his interesting inquiries to the Pathological Society on the 19th March of the present year, confirmed this discovery. Thus, it must now be admitted that tubercular matter when inoculated to rabbits invariably gives rise to the formation of tubercles, and the rule can be equally applied to guinea-pigs. When I became acquainted with Villemain's discovery I happened to be engaged with an investigation of the expectorations in phthisis, and it struck me that, if his results were correct, by inoculating the expectorations of phthisical patients, containing tubercular matter, to healthy rabbits or guinea-pigs, these animals would become tubercular; thus, I thought that a physiological

<sup>1</sup> Messrs. Herard and Cornil, Dr. Genaudet, in Paris, and M. Lebert in Germany, repeated Villemain's experiments, and obtained similar results ('Gaz. Hebdom.' January 11th, 1867).

method of diagnosis of great importance might be arrived at; for should the inoculated animals, after a certain time had elapsed, either die tubercular, or, on being killed, exhibit tubercles in any part of their body, there could be no more direct evidence as to the nature of the diseased condition of the patient whose expectorations had been used for inoculation.

If the result of the inquiry was found to be in accordance with this view, I had yet to show that the non-appearance of tubercles in guinea-pigs inoculated with expectorations from a supposed incipient case of phthisis might be considered as evidence of the patient not being tubercular. This second part of the question is more difficult to solve than the first; I am engaged with it at present. Taking into account the above considerations, it has appeared to me that the best mode of proceeding was to commence the inquiries by selecting mostly patients in the second stage of tubercular phthisis, either acute or chronic, where tubercles appear to be undergoing softening, and tubercular matter may be expected to be coughed up. Many of the patients whose expectorations were used were in-patients of the Brompton Hospital for Consumption, and I beg leave to take this opportunity of returning my most cordial thanks to the medical officers of that institution for the facilities they are kindly affording me in these and other investigations connected with phthisis.

I selected the guinea-pig as the animal for inoculation rather than the rabbit, the former being more easily obtained and convenient to keep than the latter. The inoculation was usually made in the neck and in the neighbourhood of the external genital organs, each animal being inoculated in both places. The operation was performed as follows. The sputa being collected in a capsule, a thread, passed through a needle, was thoroughly stirred about with the contents of the capsule, so as to become well impregnated with them. A small incision was then made in the skin of the animal, and the needle introduced right through the wound and under the skin, to be brought out a small distance, say a quarter of an inch,

from the incision; by means of slight pressure, with a finger, on the thread while being drawn out under the skin, the matter it contained was pressed out into the subcutaneous tissue, where it remained.

I preferred this method of inoculation to hypodermic injections with a syringe, as being less liable to kill the animal rapidly, from exerting an immediate poisonous action on the blood; and moreover a needle and thread might be renewed at each operation, thereby precluding the remotest possibility of erroneous results from the instrument not being thoroughly cleansed since last used. In a very few cases I had to use a needle over again, but then I invariably took care to heat it to redness before proceeding to operate.

I had a guinea-pig hutch made with twelve compartments, disposed vertically and closing with folding doors; these were supplied with a grating for each compartment to admit air and light; every compartment was large enough to hold four or five guinea-pigs, and was labelled on a piece of cardboard nailed to its upper partition. I took great care that the animals were properly fed. Having unfortunately no other place to keep the hutch in but a cellar, the animals were rather short of light. It might be considered that this circumstance naturally disposed them to phthisis, so that some might have become diseased independently of the inoculation; I therefore took care to keep a number of guinea-pigs not inoculated with those which had been operated upon, during the whole time the inquiry was carried on, and to kill them at the termination of the investigation for the mere sake of comparison.

Twenty-two guinea-pigs were submitted to experiment. The expectorations of nine different patients suffering from phthisis were inoculated to as many guinea-pigs; in two cases two animals were inoculated respectively with the sputa from the same patient.

Of these nine patients, eight could be safely considered in the second stage of phthisis, the tubercles undergoing softening and being expectorated.



One patient appeared to be in the chronic third stage, with cavities contracting.

The sputa of one well-diagnosed case of bronchitis was inoculated to a guinea-pig, for the sake of comparison, and one young animal of the same litter as two which were inoculated was kept without being inoculated for a similar purpose.

This will account for thirteen of the animals. The remaining nine were used in these experiments as follows:

Two guinea-pigs were inoculated with the expectorations of two cases of doubtful phthisis; one animal was inoculated with blood taken from a body during a post-mortem examination at the Brompton Hospital; another with blood procured from the last animal eleven days after the former operation. Two were inoculated with pus taken from the chest in a case of empyema, where paracentesis thoracis had been performed. Lastly, three sound guinea-pigs kept with those that had been operated upon were killed, in order to ascertain whether their internal organs were free from tubercles.

The following results were obtained from the inoculation of the eleven guinea-pigs with the expectorations from phthisical patients:—One of the animals died three days after the inoculation, obviously from some cause independent of the influence which the inoculated matter might have exerted towards the development of tubercles, and in this case no tubercles could be found in the body of the animal.

Six guinea-pigs died, and every one of them exhibited most positively tubercles to a greater or lesser extent. Of these, five died between forty-three and fifty-six days after being inoculated, and one case proved fatal in twenty-one days (it died on February the 7th), the intensely cold weather having obviously weakened the animal and contributed to the rapid termination of the disease. In the present case the guinea-pig's spleen alone was found tubercular. Four other guinea-pigs were killed at periods varying from forty-seven to fifty-four days after inoculation, and tubercles were found in every one of these animals.

There are seven more cases of inoculation to account for.

1st. Inoculation with expectorations from a patient exhibiting doubtful signs of tubercles; result—death two days after inoculation from the effects of the operation; no tubercles found.

2nd. Inoculation from another doubtful case of phthisis; animal killed fifty days after the operation—no tubercles found.

3rd. Inoculation with blood from a tubercular human dead body; animal killed eighty-three days after inoculation—tubercles found in lungs, liver, spleen, lymphatic and mesenteric glands.

4th. Inoculation with blood from last animal eleven days after the operation; guinea-pig killed seventy-two days later—no tubercles. The stage of the disease at which the blood was taken may have been too early to be productive of tubercles, hence the negative result.

5th and 6th. Inoculation of two guinea-pigs from the case of empyema; result—one animal died, tubercular, eight days, and the other was killed fifty days after inoculation. The first guinea-pig dying so soon appears to show that it was tubercular when operated upon, which does away with the value of the experiment. The second exhibited tubercles in the lungs, liver, spleen, lymphatic and mesenteric glands.

7th. Inoculation with the expectorations from the case of bronchitis; the animal was found, on being killed eighty-two days after inoculation, in no way tubercular.

Finally, not one of the three animals left sound, and which had lived with those that had been inoculated, presented on being killed and examined the slightest trace of tubercles, although they had been for about two months under precisely the same conditions as the other. And the young guinea-pig belonging to the same litter as two of the inoculated ones, which was preserved healthy, is now growing rapidly and appears in perfect health.

I may add that Mr. M. J. Salter, who assisted me in these experiments, has witnessed every one of the results which I have recorded in the present communication.

I shall now proceed to examine these cases in succession.

CASE 1.—On the 11th November, 1866, Dr. Mott, of Walton-on-Thames, requested me to see with him a young man, J. R—, æt. about 24. He had been coughing during eighteen months, but was worse for the last six months. A month previously had suffered from intestinal hæmorrhage. On the above date pulse 148; abundant perspiration; bowels loose; much expectoration; emaciation not considerable, but great prostration. Left side, anteriorly, dulness on percussion, mostly all over. Respiration cavernous at apex; very little respiratory murmur heard in that lung. Respiration natural over most of right lung anteriorly, but crackling and bronchial breathing at apex.

According to recent inquiries, I found that this man had died in the course of January last.

On the 17th or 18th November, 1866, Dr. Mott, at my request, sent me some of this man's expectorations, with which a guinea-pig was inoculated on the 19th November. I weighed the animal on three occasions till the nineteenth day after inoculation, when, finding it was continually gaining weight, I began to think the inoculation had not been effectual, and, having much other work on hand at that time, no further attention was paid to the animal. To my astonishment he was found dead on the 11th January, weighing 191·8 grammes less than when inoculated. On opening the body the state of the organs was found as follows:—In the lungs there were no tubercles; the liver was studded with tubercles; the spleen was hardened and highly tubercular, the tubercles extending deep into its tissue; the kidneys were healthy; stomach and small intestines empty; some dark matter in large intestines.

CASE 2.—Charles W—, æt. 25, an in-patient at the Brompton Hospital, under the care of Dr. Hamilton Roe. I am indebted to Dr. Dunlop, then resident clinical-assistant at the hospital, for the patient's expectorations, and he kindly favoured me with the following notes of the case:

Ill for two and a half years; on the right side cavernous sounds to third rib; posteriorly, humid crackling over upper half. On left side, submucous râles and humid crackling all over in front; posteriorly, humid crackling, with some submucous râles all over. Marked dulness over left side.

This patient died on the 2nd December, 1866. It is questionable whether a post-mortem examination was ever made, as I could find no record of it.

A guinea-pig was inoculated with the expectorations of this patient on the 22nd November, 1866, and it died on January 11th, 1867, or fifty days after the inoculation.

The condition of the internal organs was found to be as follows:—Both lungs studded with tubercles, the right rather more so than the left; no softening or cavities; spleen rough, and incipient tubercles throughout the organ; liver highly tubercular; stomach and small intestines empty.

CASE 3.—A Brompton Hospital patient, under the care of Dr. Cotton. A sample of his expectorations was inoculated to two guinea-pigs; the notes of the case, with which I was kindly supplied by Dr. Dunlop, were as follows:

William C—, æt. 28, has been ill for two years, and in the course of that time has had occasional hæmoptysis; during the last year profuse night sweats, which, however, have almost disappeared since admission. On one or two occasions has expectorated a piece of cretaceous matter about the size of a pea. The physical signs are: on right side, cavernous rhonchus anteriorly; posteriorly the respiration is harsh, without any moist sound. On the left the respiration is simply harsh, with prolonged expiration, posteriorly there is harsh respiration. This patient was discharged from the hospital on December 8th, 1866, not so well.

The first guinea-pig had 9·07 grammes (140 grains) of the expectorations injected under the skin of the neck. Immediately afterwards the animal was convulsed, and the next morning it felt cold and remained very quiet. It died

on the evening of November 6th, or two days after the inoculation. There was serous effusion in the subcutaneous cellular tissue. I could find no tubercles in the internal organs.<sup>1</sup>

Six minims of the same patient's expectorations were injected with a hypodermic syringe under the skin of the second animal on December 5th, 1866. On January 11th he appeared very sickly; on the 15th a little blood was taken from it, and inoculated into the neck of a small guinea-pig, which died two days afterwards, when no tubercles were found in its body.

The animal injected with the expectorations was observed on the 15th January to be apparently hungry, nibbling readily at its food, but actually eating very little; it died two days later, or forty-three days after inoculation, and was examined immediately after death. There were no obvious tubercles in the lungs; the liver had a large tubercular mass perforating its substance through and through, and was very much softened about that spot. The stomach was full of frothy fluid, very alkaline; kidneys healthy. I can find no record of the state of the spleen.

CASE 5.—S—, æt. 32, much emaciated; hereditary disposition to phthisis from mother very doubtful, absent on father's side. He has had two attacks of hæmoptysis in the last seven years. Been subject to a cough all his life. Became suddenly worse five months ago, after spitting blood. At present much dyspnœa when moving about; perspires occasionally at night; expectorations abundant; percussion note somewhat dull all over both sides. On auscultation, anteriorly on both sides, but very little respiratory murmur is heard. Some crackling sounds at places; no very obvious cavernous respiration. Some normal respiratory murmur heard posteriorly.

On January 12th six minims of the expectorations of this

<sup>1</sup> My notes only refer in this case to the lungs and liver, where no tubercles were found. It is very obvious that the animal died from some alteration of the condition of the blood due to the injection.

patient were injected under the skin of a guinea-pig with the hypodermic syringe. On the 26th an abscess had formed at the spot of injection, which was opened, and much fetid pus issued. On February 12th granulations had formed near the wound, having a tubercular aspect.

It died February 28th, or forty-seven days after inoculation. The animal at first gained in weight, and then fell off again, being when he died a little heavier than when inoculated.

The appearance of the body after death was as follows :—Left lung studded with tubercles, more especially in the upper lobes, two clusters of tubercular masses running into each other. Right lungs also studded with tubercles throughout, many feeling very hard on pressure; liver exhibits a number of small tubercles. Two hard tubercular masses in pancreas; a few in the mesenteric glands; spleen large and also tubercular; kidneys healthy. Stomach contains a green fluid reacting alkaline; small intestines nearly empty; the large intestines contain some fæcal matter.

CASE 6.—Jane N—, æt. 25, under Dr. Cotton, at the Brompton Hospital. I am indebted to Dr. Aitken, resident clinical-assistant at the hospital, for the following notes of the case :—One brother died of phthisis. Ill since autumn 1865. Cough only slight up to Christmas of that year; worse since that time. On admission expectorations mucopurulent, mixed with blood. Catamenia very irregular, but present at last three periods. Leucorrhœa; great debility.

Physical signs on the 15th December.—On left side dulness, amphoric respiration; infra-clavicular gurgle. On right side respiration amphoric. The patient was discharged on February 20th, much improved.

On the 16th February a guinea-pig was inoculated with the expectorations of this patient. I performed the operation in the neck and near the external genital organs, a needle and thread being used for that purpose. The animal had been in my possession about a month, and was then in good health. On the 21st March three small ulcerations appeared

at the spot of inoculation near perineum, and also scars at the back of the ear.

On the 9th April, or fifty-two days after inoculation, the animal was killed and his body examined. The lungs were healthy. In the liver a tubercular mass about the size of a large duck-shot. Very distinct incipient tubercles in spleen. One mesenteric gland enlarged to the size of a small French bean and indurated, but contains no softened cheesy matter.

CASE 7.—This was a patient named John S—, under the care of Dr. Hamilton Roe, at the Brompton Hospital. Dr. Aitken kindly supplied me with the following notes of the case:—On the left side exaggerated respiration. On the right, flattening below clavicle, humid crackling only in axilla and at isolated points; cavernous respiration limited and close to sternum. Posteriorly no sibilus, respiration weak. No comparative dulness between percussion note on both sides. He left the Hospital on March 23rd, some sibilus being then heard in the right side, and on the left side some crepitation below third rib and in axilla.

A healthy guinea-pig, which had been kept in the hutch about a month, was inoculated with the expectorations from this patient on February 16th; the inoculation was made in two places, as in the former case, with needle and thread. On the 21st March a deep ulceration was observed at the place of inoculation near perineum. The animal was killed on the 8th April, or fifty-one days after inoculation.

The lungs contained no tubercles. In the liver there were two tubercular masses (the substance of which exhibited very indistinct cellular structure under the microscope). Mesenteric glands hardened, but not cheesy; spleen covered with tubercles newly formed; kidneys healthy; stomach full of green food.

CASE 8.—Margaret C—, æt. 22. A patient of Dr. Roe, at the Brompton Hospital. I am also indebted to Dr. Aitken for the notes of the case.

The physical signs on the 29th January were: left side

apex loud humid crackling, respiration harsh; bronchial cough, and comparative dulness and flattening below clavicle and limited motion on that side. At base of left side posteriorly, sub-crepitant rhoncus. On the right side, no signs but occasional moist crackling. She left the hospital, relieved, on March 2nd, when no crackling sounds were heard on right side.

In the present case the guinea-pig was inoculated with the expectorations of the patient in the back of neck only, the operation being performed on January 17th; it was found dead on March the 15th or fifty-six days after the inoculation. On opening of the body one tubercular mass was found at apex of right lung, and four on the posterior surface of lower right lobe, with some very small ones just commencing. On a close inspection of posterior surface of left lung one minute tubercle detected just beginning. The tubercles on right lung show well the incipient stage of formation; the first sign is a small speck just visible on the surface of the lung, exhibiting a grey homogeneous colour; this gradually enlarges (in the present specimen it has attained one eighth inch in diameter), retaining its grey homogeneous appearance, but the largest of these masses has a white speck in the centre. There are two tubercular masses in the liver; spleen covered with incipient tubercles; the stomach contains some alkaline fluid and no food. The duodenum near stomach empty, but about three inches from the pilor some food in process of absorption.

CASE 9.—The same patient as the last one. The physical signs were then much the same as about a month previously, when the first animal had been inoculated with this patient's expectorations.

The guinea-pig used on the present occasion had been under observation for above a month, and was in good health; on February 16th he was inoculated by means of a needle and thread, as on the former occasion. The animal was killed on the 8th April, or fifty-one days after inoculation.

The lungs were found to be free from tubercles. On close



examination of the liver five or six distinct small tubercular masses were seen on its surface. Spleen much enlarged and covered with tubercles; it weighed 2·149 grammes (average weight of three healthy spleens 0·785 grammes), felt hard at places, but not throughout. One mesenteric gland hardened though not much enlarged; on cutting into it, a soft cheesy mass can be pressed out, showing, under the microscope, small granular irregular cells.

The spleen was sent to Mr. Tuson, who made me a coloured drawing of it. (See Plate III, fig. 1.)

CASE 10.—Bessie S—, Brompton Hospital, under the care of Dr. Pollock. (Chronic advanced second stage.)

On both sides humid crackling. No comparative dulness, harsh respiration.

Left hospital, improved, on January the 24th.

The guinea-pig—a young one of the same litter as that used in the case of Margaret C—, was inoculated in the back of neck with needle and thread on January 17th, and died on February 7th, or twenty-one days after inoculation. In this case the lungs and liver were found healthy, but there were tubercles in the spleen.

With the object of ascertaining the influence of the inoculation on the growth of the inoculated guinea-pigs in both the cases of Margaret C— and Bessie S—, three young animals had been selected belonging to the same litter, and whose mother was also in my possession. Two of the young guinea-pigs were inoculated respectively with each of the above patients' expectorations, while the third was kept in a sound condition for the sake of comparison.

As time elapsed the weights of the three animals varied as follows:

	Margaret C—.	Bessie S—.	Healthy animal.
	Grammes.	Grammes.	Grammes.
Weight before inoculation	103·25	95·17	
5th day after	112·82	104·82	
7th     "     "	127·01	123·2	158·85
9th     "     "	135·25	127·5	167·85
13th   "     "	149·1	141·6	181·6
16th   "     "	150·0	141·25	177·6
21st   "     "	157·6	147·5 (died)	198·5
26th   "     "	168·8	.....	211·43
27th   "     "	161·5	.....	207·1
30th   "     "	170·24	.....	238·65
33rd   "     "	165·65	.....	230·4
36th   "     "	160·06	.....	216·11
39th   "     "	173·4	.....	241·5
42nd   "     "	178·1	.....	245·3
46th   "     "	177·85	.....	263·0
48th   "     "	182·6	.....	—
51st   "     "	182·5	.....	261·12
54th   "     "	173·8	.....	272·0
57th   "     "	died	.....	—

1st. The animal inoculated with the expectorations of Margaret C— had gained from the seventh day after inoculation till death 46·79 grammes, while the healthy animal during the same time had gained 113·15 grammes, or nearly three times as much.

2nd. The guinea-pig inoculated from Bessie S—, from the seventh to the twenty-first day increased by 24·3 grammes, while the healthy one during the same short period gained 39·65 grammes.

The mother and sound guinea-pig were still in my possession when this paper was sent in, both apparently quite healthy.

CASE 11.—H. P—, under my own care. In December, 1865, hæmoptysis to about two pints. No hæmorrhage since then but occasional rusty sputa, and expectorations purulent as ascertained by the microscope. Coughs a good deal, has been taken worse since the beginning of January last when the cold was so acute.

Physical signs on the 27th February, 1867. Left side anteriorly: bronchial breathing; expiration noisy and pro-

longed, now and then friction sounds; no gurgling. Lower down, complete, or nearly total, absence of sounds of any kind. Expansion deficient; percussion note on that side anteriorly quite dull throughout.

Left side posteriorly: loud crackling near apex at end of noisy expiration; both inspiration and expiration bronchial; some distant and indistinct respiratory murmur. Lower down, distant and coarse respiratory murmur. Percussion note dull at apex, tolerably resonant elsewhere.

Right side anteriorly: at apex, deficient respiratory murmur with crackling: lower down, respiration heard with sibilus. Posteriorly: at apex, respiration coarse; elsewhere tolerably healthy with sibilus. On percussion, resonant throughout.

A guinea-pig was inoculated, as usual, with the expectorations of this patient on March 7th, and killed on April 24th, or forty-eight days after the operation. There was an ulceration in the neck, where it had been inoculated, and near genital organs an abscess had formed at the inoculated spot. The contents of this abscess being submitted to microscopical examination were found to have the appearance of pus. The state of the internal organs was as follows:—Lungs quite healthy, but a little serous fluid in the thoracic cavity; liver healthy; spleen studded with tubercular masses. (See Plate III, fig. 2.) These had the form of hard, little, round, yellowish bodies, somewhat like small shot; when cut through and examined with the microscope, they were found to contain mostly cells bearing some analogy to pus, but on a close examination, smaller and irregular cells, with free granular matter, were found. Some of the cervical lymphatic glands swollen and considerably indurated; on being cut into, scarcely any softening was found.

CASE 12.—Annis D.— This case—one of doubtful phthisis—is in great measure devoid of interest, from the inoculated animal having died two days after the operation, obviously from serous effusion throughout the body. None of its organs could be found to exhibit any tubercles.

CASE 13.—Bronchitis. John K—. This was a genuine bronchitis case, the expectorations of which were procured at the Brompton Hospital, by Dr. Dunlop.

I inoculated them, with every care, to a healthy guinea-pig, in the neck and near genital organs, on the 17th January, 1867.

It is worth noticing that the weight of this animal increased regularly from 396 grammes to 561 grammes in the course of eighty-two days, which elapsed between the date of inoculation and the 9th April, when it was killed, being apparently in perfect health. The lungs, liver, spleen, pancreas, kidneys, and mesenteric glands were found healthy. There were on the under surface of the liver a few small white specks, not unlike small grains of sand in size, and loosely scattered over the surface; they could be removed with the end of a knife, and when submitted to the microscope were found to consist of large round or oval granulated cells, with a well-defined regular margin and about three times the diameter of the usual tubercle cell; they were also considerably larger than pus-cells.

I can merely add that in other guinea-pigs, not inoculated, and in every respect healthy, the same appearance in the liver was found, and I cannot believe it to be in any way inconsistent with health.

CASE 14.—Lydia R—, under the care of Dr. Alison, at the Brompton Hospital. I obtained the expectorations through the kindness of Dr. Evans, Resident Clinical-Assistant. This was a doubtful case of phthisis with but slight expectoration.

A guinea-pig was inoculated with this patient's sputa, on March 5th, the operation being performed with the usual care in the neck and near the genitals. It was killed on April 24th, or fifty days after the inoculation, when the lungs, liver, spleen, kidneys, and mesenteric glands were found quite healthy.

The patient was discharged, on April 16th, in fair health.

CASE 15.—On the 15th January I took some blood from the body of a patient who had died at the Brompton Hospital on the 12th. The post-mortem examination of this patient showed her right lung to be highly tubercular, with cavities and consolidation of tissue. The blood was taken directly from the superior vena cava.

A healthy guinea-pig was inoculated with this blood in the usual way by means of a needle and thread. The weight of the animal—318·2 grammes—first rose to 421 on the twenty-third day after inoculation, and then fell to 364 on April, or eighty-three days after inoculation, when it was killed.<sup>1</sup>

The state of the organs was found to be as follows:—

There was a circular ulceration in the neck, probably at the point of inoculation. The lungs on the right side exhibited tubercular masses very hard on pressure, about two thirds of the entire lung on that side being unfit for respiration. The left lung contained a few fresh tubercular masses. On the liver there were four small tubercular deposits, and one large one the size of a maize grain. A cervical lymphatic gland, on left side, was white, hard, and enlarged to the size of a marble. Mesenteric glands but little affected. Spleen hardened and covered with tubercles. The contents of the lymphatic gland resembled soft white paste, and exhibited, under the microscope, very small, irregular granular corpuscles.

CASE 16.—Blood from this last guinea-pig, taken eleven days after inoculation, was inoculated on January 26th to a healthy guinea-pig, which apparently remained in perfect health. On April 8th, or after seventy-two days, it was killed, when no tubercles were found in its body. Small sand-like, white particles, as those previously referred to, existed in its liver. The mesenteric glands were rather indurated, but contained no white, cheesy, tubercular matter,

<sup>1</sup> Nine days after inoculation the cornea of this animal's left eye was observed to have become opaque from a deposit of lymph. Two days afterwards the other eye was affected in the same way, and from that time sight must have been very much impaired, if not entirely destroyed.

CASES 17 and 18.—This was a very interesting instance of empyema I saw in consultation with Dr. Mott, of Walton-on-Thames.

It is not my intention, on the present occasion, to enter at all fully into the particulars of this case. The patient was a child, then aged five years, on whom the operation of paracentesis thoracis was performed by Dr. Mott, and during sixteen months a purulent fluid continued on and off to pour out through the opening. From the history of the case, and general symptoms, the child appeared tubercular; auscultation and percussion yielded but little information on that point. In order to solve the question, I obtained some of the pus, with which two guinea-pigs were inoculated on March the 4th, one of them by the usual process, and the other by injection with a hypodermic syringe, two minims of the pus being introduced under the skin of the abdomen.

The child died on the 22nd March, his urine having become for some days before death very scanty and albuminous.

The body was examined by Dr. Mott, who informs me that he did not find any tubercles present. It is difficult, however, to reconcile the case with an hereditary predisposition to phthisis, its progress and symptoms and the very marked club-shape of the boy's fingers, without a strong suspicion of the existence of the tubercular virus (if we admit Villemin's views), in the patient's body; this virus might have been in progress of elimination with the discharge, which would account for the formation of tubercles in the inoculated guinea-pig, and in some measure for the absence of the development of tubercles in the patient's body.

The animal inoculated with needle and thread died eight days after the inoculation, having consequently lived so short a time subsequently to the operation that the diagnostic results obtained from the state of its body can hardly be accepted. The animal's liver was studded with tubercular masses (see Plate III, fig. 3). The spleen and mesenteric glands were also tubercular. The lungs and kidneys were healthy.

The second guinea-pig, which was inoculated by injection, was killed on April 24th, or fifty days after the operation.

The lungs were both found to contain a few grey, translucent tubercular masses in the very earliest stage of formation, also on pressing the lungs between the fingers small hard masses could be distinctly felt like minute pellets of shot. The spleen was also infiltrated with tubercles. The tubercular masses could be cut out of this organ, when they appear as yellowish round bodies; on pressing them between two microscope slides they burst, emitting the usual white pulpy substance seen under the microscope to consist of small, irregular, granular cells, with much free granular matter, these being apparently the characters of tubercular matter. One small tubercular mass was found in the liver. Several mesenteric glands were very much enlarged and hardened, containing a white pulpy substance very like the sebaceous matter from acne, and under the microscope found to consist of small irregular, granular cells, with very little free granular matter. There were a number of small tubercular masses in the diaphragm, the contents of which were apparently, under the microscope, highly characteristic of tubercle, and also some tubercular lymphatic glands.

Finally, as stated at the beginning of this communication, in order to make sure that the hygienic conditions under which the animals were kept, had in no way been the cause of the formation of tubercles in any of the cases described above; three guinea-pigs which had throughout been kept along with the others, and consequently exposed to the same influences respecting food, air, and light, were killed on April 9th, and their bodies carefully examined, when no tubercles were found in any of these animals.

In conclusion, I believe the results of the experiments and observations related in the present paper to be as follows:—

1st. The inoculation of guinea-pigs with the expectorations of patients suffering from tubercular phthisis will, at all events in a certain stage of the disease, and possibly throughout, give rise to the formation of tubercles in the operated animals.

Plate III

Fig. 1.



Fig. 2.



Fig. 3.



Fig. 4.





2nd. If two or more guinea-pigs inoculated with human expectorations, brought up by coughing, should die from tubercular disease, or should, on being killed at least thirty days after inoculation, exhibit tubercles in their body, this may be considered as a direct and positive evidence that the person whose expectorations were inoculated was suffering at the time from tubercular phthisis.

3rd. If two or more guinea-pigs be inoculated with the expectorations coughed up by a person considered to be in the third stage of phthisis, and if these animals do not die of tubercular disease, or exhibit any tubercles when killed, at least fifty days after inoculation; it may be considered that, in the present case, the softening of tubercles and the secretion from the pulmonary cavities are arrested, the patient being in a fair way of recovery.

4th. Besides the pulmonary expectorations, blood and pus taken from the human body in phthisis appear to be also possessed of the power of causing the formation of tubercles in guinea-pigs, when inoculated to these animals.

5th. The spleen appears to be the first, and the lungs one of the last organs, in guinea-pigs, to be attacked with tubercular disease.

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#### DESCRIPTION OF PLATE III.

Fig. 1.—Tubercular spleen from a guinea-pig, Case 9. Incipient condition.

Fig. 2.—Tubercular spleen from a guinea-pig, Case 11. Advanced state.

Fig. 3.—Tubercular liver from a guinea-pig (tubercles probably not from inoculation).

Fig. 4.—Healthy spleen from a guinea-pig.

Plate III

Fig. 1



Fig. 2



Fig. 3



Fig. 4



11

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CONTRIBUTIONS  
TO THE  
PATHOLOGY OF ANEURISMS AND  
TUMOURS,  
INVOLVING THE UPPER PORTION OF THE CHEST  
AND ROOT OF THE NECK.

BY  
JOHN COCKLE, M.D.

COMMUNICATED BY  
F. SIBSON, M.D., F.R.S.

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THE following examples of aneurismal and other tumours involving the upper part of the chest and root of the neck, are submitted to the notice of the Society as exhibiting points of interest in the pathology and diagnosis of these affections.

The first case is one of traumatic aneurism of the innominate artery, with eventually either sudden yielding or consecutive rupture of the sac. (See Plate IV, fig. 1).

John V—, æt. 35, a cabinet-maker by occupation, was admitted into the Royal Free Hospital under the author's care, June 12th, 1865, with the following history :

He had enjoyed tolerably good health until eighteen

months ago, when he fell with great violence upon the right shoulder. This injury compelled him to keep his arm in a sling for a period of three weeks. No further complaint was made until five or six weeks ago, when he felt pain in both shoulders, but more severe in the right than in the left shoulder. A fortnight previous to his admission he observed, on rising in the morning, that his throat was swollen. When admitted, the sternal end of the right clavicle was observed to be displaced forwards by a pulsating tumour extending from between the heads of the sterno-mastoid muscle into the episternal notch, and forming in this situation a considerable globular prominence (the size of a hen's egg). The impulse of the tumour was circumscribed, forcible, expansile, and highly liquid over the entire surface during the diastole of the artery, with a faint recoil during its systole. Slight silvery fremitus was felt over the swelling during each dilatation of the artery. Marked concussion impulse, and a loud and somewhat harsh arterial diastolic murmur were observed over the swelling and at the sterno-clavicular articulation. At this region the percussion sound was dull, but both dulness and murmur diminished sensibly below these limits. The murmur was transmitted into the right carotid and subclavian arteries. The contents of the tumour could be partially displaced by pressure. The trachea appeared to be pushed somewhat to the left side; the voice was harsh and metallic; the right radial pulse was weaker than the left; the right carotid and temporal arteries were also weakened in pulsation, but not to the same extent; pressure on the right carotid artery sensibly diminished the pulsation in the tumour; the superficial thoracic veins were large and turgid. The right conjunctiva was suffused, but there was no inequality in respect of size of the pupils; heart and lungs apparently healthy. June 17th.—Impulse in tumour much diminished; the tumour appears larger but flatter upon the surface; the lateral expansion increased; the superficial veins are smaller; pulse 88; respirations 20 per minute.

20th.—Has suffered from troublesome cough the last two nights with slight expectoration of frothy mucus. The axillary artery was compressed for an hour and a quarter, and the carotid artery for fifteen minutes, which was as long as the compression could be borne.

21st.—Pulsation in tumour slightly diminished; cough less frequent during the night; pulse 80; respirations 20; harsh voice absent; trachea in normal situation; compression of the axillary artery for upwards of an hour; carotid artery three quarters of an hour.

22nd.—Passed a good night; cough better; right radial pulse weaker; 76 pulsations per minute; respirations 24; impulse less over sac and slightly less liquid; pressure applied to both vessels for one hour and ten minutes.

23rd.—Bad night; complains of pain in the right cheek and in the infra-scapular region; larynx displaced to the left side; voice harsh and laryngeal; sharp, ringing cough; no difference in the size of the pupils; impulse over sac faint both during diastole and systole of the artery.

24th.—Impulse in tumour less; pressure applied to the arteries for an hour.

25th.—Tumour appears much smaller with still diminished impulse; pulse and respiration normal; pressure on the arteries reapplied.

27th.—Headache; impulse less; pressure on the arteries daily for one hour.

30th.—Impulse less, scarcely visible; the tumour feels firmer to the touch.

The case seemed to improve up to—

July 7th.—At this date pain at the back of the head was complained of, particularly after pressure upon the vessels.

9th.—Headache; a sulcus can now be seen bisecting the tumour vertically, which latter feels more solid to the touch.

11th.—Severe headache; cough troublesome at night; the tumour has become more prominent. From this date to—

21st.—The prominent symptoms were severe pains

in the head, shoulders, chest, and back ; on this day additionally, pain in the sternal region immediately below the margin of the tumour, which latter has now extended to the left sterno-mastoid muscle.

23rd.—Tumour larger ; impulse increased ; right radial pulse absent ; pain in the right arm extending from the tumour down to the elbow.

August 1st.—Severe pain in the shoulder preventing rest ; the tumour presents the appearance of a cricket-ball.

4th.—Pain in the right arm as far as the wrist with occasional numbness ; right pupil slightly contracted.

6th.—Bad night : feeling of pressure on the windpipe, causing a sense of suffocation ; headache and cramp in the arm, to—

11th.—No change beyond enlargement of the superficial veins of the forearm ; puffiness of the shoulder ; loss of feeling in the little, and half of the ring finger.

15th.—Physical phenomena the same ; slight clubbing of the fingers ; right pupil still smaller than the left ; some difficulty in swallowing.

16th.—Superficial veins of the right arm more developed ; right palpebra turgid and inflamed.

19th.—Temperature of right hand and arm considerably above that of the left.

21st.—Temperature of right arm higher by  $10^{\circ}$  than that of the left.

25th.—Pulsation in sac nearly synchronous with left radial pulse ; feeble pulsation in right brachial and ulnar arteries ; radial pulsation absent ; right conjunctiva red and lachrymose ; palpebra swollen ; ulceration at right outer canthus ; pupil contracted.

27th, at 10 a.m.—Sudden and intense dyspnœa ; pain in neck and harsh cough ; said “ he felt as if the tumour had burst ; ” pulsation entirely absent at the right wrist and also in the sac itself ; tumour greatly diffused towards both right shoulder and left sterno-mastoid muscle. (See Plate IV, fig. 2.)

28th.—Tumour larger and more diffused, extending to

the left side of neck and right shoulder ; three attacks of suffocative dyspnœa.

29th.—Pulsation returned in sac ; general engorgement of the lower lip.


September 1st.—Left radial pulse small ; great extension of swelling round the neck ; face swollen and pallid ; considerable dysphagia ; food obliged to be cut very small. (See Plate IV, fig. 3.)

5th.—Tumour extending daily and becoming more diffused ; ecchymosis of skin anteriorly ; redness, heat, and tension at the lower portion of the tumour. (See Plate IV, fig 4.)

8th.—Died suddenly. Within five minutes after death the immense tumour had completely collapsed, leaving a slight depression, with an elevated ridge at its lower portion. (See Plate IV, fig. 5.)

Post-mortem examination two hours after death :—The sac collapsed as described, the sternal end of the right clavicle displaced forwards, larynx and trachea displaced an inch and a half to the left side ; congestion of the external mammary veins. The right pupil more dilated than the left, whereas, before death, the right pupil was most contracted. An incision was carried from the chin to the symphysis pubis, dividing the skin and adipose tissue of the chest, and laying open the abdominal cavity ; a second incision extended from the chin to the mastoid process of the temporal bone, and a third incision from the sternum to the acromion process of the scapula ; the integument was separated from the muscles on either side of the neck and chest in order to expose the relations of the aneurism. The connective tissue between the integument and pectoral muscles exhibited a brawny condition. Upon dissecting away the pectoral muscle on the right side, the tissue was dense and firm, resisting the edge of the scalpel ; the muscles were pale and atrophied compared with those of the opposite side ; the external and anterior jugular veins on this side were pervious ; the sterno-mastoid muscle was bulging and prominent, stretched over the sac, but pale and wasted



compared with that of the opposite side. The ribs on the left side were now cut through, the right clavicle, with first and second ribs, were divided on a line drawn from junction of the middle with the outer third of the clavicle, and turned forwards. The sternum was next cut through; the pericardium and lungs having been exposed, the subclavian muscle, vein, and artery were now seen with the upper part of the pleura: these parts formed the anterior and inferior coverings of the sac. The sterno-mastoid muscle was next divided and found intimately adherent to the sac, and, with the cervical fascia, formed its anterior and superior coverings. Tracing the sac to the left, it extended an inch and a half beyond the mesian line, displacing the œsophagus; inferiorly, it was bounded by the apex of the right pleura; anteriorly, stretching the sterno-mastoid muscle and eroding the sternal end of the clavicle which was pushed forward one inch more than that of the left side; superiorly, it extended upwards on a level with the thyroid cartilage; externally, it extended as far as the trapezius muscle, having the cervical fascia, considerably thickened, for its chief covering; posteriorly and internally the sac was bounded by the common carotid artery, internal jugular vein, pneumogastric and sympathetic nerves, trachea, larynx, and œsophagus, all of which organs were compressed and displaced towards the left side. An aperture of somewhat quadrangular shape  exists in the anterior wall of the innominate artery, with this exception, the artery is perfectly healthy throughout its entire extent. Neither does the slightest disease exist in any other part of the large vessels or in the heart itself. No appearance of any former injury of the right clavicle is discernible.

CASE 2.—This case (see Plate V, fig. 1) most strikingly resembles that last described, so far as relates to the exact position of the pulsating tumour in its earliest stage, but differs from it signally in its spontaneous origin, and in being most probably one of true aneurism or dilatation of

the ascending and origin of the transverse aorta, innominate and root of the right common carotid arteries.

Mary Ann E—, æt. 52, a married woman, was admitted into the Royal Free Hospital under the author's care, September 12th, 1866, with the following history:—She had always enjoyed good health until twelve months previous to her admission, when she first felt a pulsating swelling in the throat (episternal notch). This swelling came on gradually, without any obvious cause, and was soon followed by dimness of vision in the right eye; the only remaining symptoms complained of have been pain, at times, near the top of the sternum, and an occasional sense of choking; the pain complained of seemed, in part, external, and the tissues, some time since, felt somewhat thickened over the region of the right sterno-clavicular articulation and upper part of the sternum, and still remain tender even under the gentlest percussion. The tumour appears to arise near the former region, encroaching upon the outer head of the right sterno-mastoid muscle and passing into and filling the hollow above the episternal notch. The origin of the right common carotid can be felt dilated. The tumour has varied considerably in point of visible size; some weeks ago, about the period of admission, it was much more prominent than at the present time, bulging out as large as a hen's egg from the surface. The impulse has invariably been forcible, markedly expansile, and very liquid, with slight thrill (diastolic arterial) limited to the space between the heads of the right sterno-mastoid muscle. Perfect collapse of swelling during systole. A loud and somewhat harsh murmur is audible over the tumour during the arterial diastole, and, though much weaker, during the systole; it passes loudly during arterial diastole into the right carotid, subclavian, and brachial arteries, and is even faintly heard in the arteries of the left side. The double murmur, well marked, passes down the aorta, but is most decidedly weakened near the aortic mouth and at the apex of the heart. The pulsation in the carotid arteries has been, as a rule, equal, but a strongly marked difference has existed in the right radial pulse, which

is at times full and strong, almost jerking in character, at other times very sensibly weakened, as is well shown in the sphygmographic tracings taken at various times by Dr. Anstie, Dr. Sibson, and the author. There is some dulness on percussion over the upper portion of the sternum, but no tactile or stethoscopic impulse can be detected. The position of the heart, its sounds, and impulse, are normal; the lungs also appear in a physiological condition. This patient has just quitted the wards of the hospital (June 23rd), her condition being unchanged.

**CASE 3.**—This case is an interesting complement to the last, being one, probably, of more general dilatation of the aorta, and also involving the innominate trunk and origin of right carotid artery.

Emma R—, æt. 46, married, has been an out-patient of the Royal Free Hospital, under the author's care, for nearly five years. She first complained of swelling and pulsation in the hollow of the throat, speedily followed by dyspnoea, pain in the chest, and very troublesome cough. An inter-mitting prominence is visible at the lower part of the episternal notch, the swelling becomes very marked during the arterial diastole, but quickly emptying itself during systole; the swelling extends more definitely from behind the right sterno-clavicular articulation along the tracheal border of the sterno-mastoid muscle, where the arterial enlargement is readily traceable; abnormal pulsation existed, though in a less degree, in both carotid and subclavian arteries. In the earlier stage neither thrill nor murmur could be detected; the pulsation of both radial arteries was comparatively weak: the second cardiac sound was unusually sharp and clear. At a later period, in the progress of the case, the pulsation in both radial arteries became somewhat jerking in character, and a double murmur, louder and harsher during arterial diastole became audible down the aorta, extending to the apex of the heart; the impulse of this organ being increased, and signs of hypertrophy of the left ventricle being superadded. Marked percussion dulness

exists at the right sterno-clavicular junction and down the entire course of the ascending aorta. The tracings attached were taken some weeks ago by Dr. Anstie. The left jugular vein is nearly always in a state of permanent distension, and presents, at times, visible pulsations. Dyspnœa and constant cough are permanent.

CASE 4.—*Vast aneurism of the aorta, with secondary sacculation ascending above the sternal end of the left clavicle over the site of the carotid artery.* (See Plate V, figs. 2 and 3).

Deborah W—, æt. 36, sempstress by occupation, married, has had four children, and one miscarriage. She was admitted an out-patient of the Royal Free Hospital, October 19th, 1866. Ten years ago she contracted syphilis from her husband, and since that time has been so maltreated by him that she thinks her chest may have sustained some injury from his violence. During the last two years she has lost flesh very considerably, otherwise she had previously enjoyed tolerably good health with the exception of cough and cold during the winter months. Nine months prior to her admission to this hospital she began to feel pain in the region of the left sterno-clavicular articulation, which pain she considered to be rheumatic, and for which she thinks she was treated at University College Hospital, where she became a patient. This pain she described as being, at times, intolerable. About this period a gradually increasing hoarseness occurred, uninfluenced by either local or constitutional treatment. Three months later she observed a swelling in the situation of the articulation named. When first seen here, a firm, unyielding tumour was detected in the neighbourhood of the left sterno-clavicular articulation and inner third of the clavicle, pushing upwards and forwards the clavicle and clavicular attachment of the sterno-cleido-mastoid muscle, the inner third of the clavicle being lost, as it were, in the tumour, the whole mass conveying the impression of an osteoid growth. No impulse existed over the swelling, but a double sound could be heard through the tumour as

if communicated by the common carotid artery over which the tumour seemed to be immediately lying. At this time, iodide of potassium was administered internally, and an opiate lotion applied over the swelling.

October 26th.—Seven days afterwards the tumour was smaller in size and less painful; the patient expresses herself relieved, but there is no apparent change in respect of density of the tumour to the touch.

November 15th.—About three weeks subsequently she was transferred to the author's care. She was now greatly emaciated, with a marked leaden tint of complexion and livid lips. The breathing was quickened, although she made no complaint of dyspnœa. Indeed, her only complaint was of pain in the old spot, weakness of her voice, and occasionally double vision. A tumour was to be seen, about the size of a small hen's egg, displacing the left sterno-clavicular articulation, and passing up behind the inner third of the clavicle beneath the origin of the sterno-mastoid muscle, encroaching somewhat upon the hollow of the throat, and extending about an inch and a half up the neck in the track of the carotid artery, slightly displacing the larynx to the right. A very slight visible impulse existed in the tumour; the skin covering it was ecchymosed with a congested state of the capillaries surrounding it. The veins below the clavicle, more particularly upon the left side, were distended and tortuous. No bulging of the general surface of the chest was perceptible, beyond a fulness at and below the left sterno-clavicular articulation corresponding to about the second intercostal space. At this stage of the examination the impression was conveyed of the case being aneurism of the carotid artery. Upon further examination, however, the tumour seemed to fuse into the substance of the clavicle itself, whilst the faint impulse, almost limited to the apex of the tumour, felt very liquid; the apex itself was readily depressed with the point of the finger; no murmur whatever could be detected over the swelling. The upper third of the entire chest, anteriorly and posteriorly, was uniformly dull upon percussion, with a marked sense of tactile resistance.

No unnatural impulse whatever could be detected by the hand firmly placed over the area of dulness. Below this area, the percussion sound was clearer, but yielded by no means a natural resonance. The area of the heart's impulse was increased; the impulse was forcible and jerking, and the cardiac sounds muffled and indistinct, but without murmur or thrill. The circulation was very feeble, entirely absent in the left radial and ulnar arteries, and exceedingly feeble in the right radial artery. All respiratory murmur was extinct over the upper portion of the chest, but over the remaining part slight dry, and liquid râles were audible. At the root of the neck, on either side, an impulse existed, one of simple elevation, without either thrill or murmur, as if some very solid body were heaved up at each contraction of the heart. At this stage of the case great doubt was entertained whether it was one of intra-thoracic tumour, or a large aortic aneurism filled with solid clot; the latter conclusion was eventually drawn, based principally upon the peculiar elevation on either side of the root of the neck during each contraction of the heart, and the general condition of the circulation.

After some short interval (two or three weeks) the following changes occurred:—The tumour in the neck subsided, completely, permanently, and with comparative quickness. In proportion to its disappearance, the external chest veins increased in size and became more tortuous and, now, pulsating. (See Plate V, fig. 3.) Pulsation could no longer be felt in the right radial, carotid, and temporal arteries. When the patient was placed recumbent, faint pulsation was to be felt in the abdominal aorta and crural arteries. The only change in the symptoms was an increase in the frequency of double vision, contraction of the left pupil, transient loss of power in the limbs, and occasional momentary unconsciousness on awaking in the night. The patient (refusing constantly to be admitted into the wards) managed, in this condition, to walk to the hospital twice in the week, for a few weeks. No pulsation could ever be detected at the wrists, although frequently examined by

different observers. She fell suddenly dead while in the act of preparing some vegetables for dinner—a not frequent example of aortic aneurism terminating suddenly, without rupture of the sac or more than ordinary exhaustion. The death was, probably, syncopal. The author is indebted to Mr. J. D. Hill for the account of the post-mortem examination. The entire specimen is preserved, but the sac has not been laid open.

*Post-mortem examination twenty-four hours after death.*—

The body was greatly emaciated, and exhibited bruises upon the face and nose. No tumour was visible externally. When the chest and abdomen were laid open, an immense aneurismal tumour occupied the upper half of the left side of the thorax, encroaching upon the upper and inner half of the right side. The sac was adherent, in front, to the under surface of the left clavicle, first rib, and intercostal muscles, and, posteriorly, to the contents of the posterior mediastinum by dense areolar tissue. Upon the right side the sac encroaches upon the right lung, being adherent to the pleura, and drawing the pericardial layer of pleura, resting upon the root of the lung, and compressing the bronchial vessels and tubes. Upon the left side the sac crowds the lung into the outer half of the chest; it is adherent to both layers of pleura, and presses upon the bronchial tubes and vessels. The lung is quite solid, and adherent at its apex to the upper part of ribs and sac. Superiorly, the sac is still seen above the clavicle; the contents of the posterior mediastinum are compressed, and pushed out of their normal position. The trachea is dislocated two inches to the right side: the œsophagus is also displaced, though to a less extent. Inferiorly the sac is attached to the pericardium, and displaces the heart downwards, the apex of this organ corresponding to the seventh intercostal space two inches beyond the costal attachment of the cartilages. The right lung was condensed at its base, and adherent to the sac. The left subclavian artery is very much smaller than the right. Atheromatous disease, in an early stage, exists in the right carotid

artery. The left innominate vein is obliterated, and its place probably supplied by a branch of the thyroid vein coursing across the front of the sac between the sternomastoid and sterno-hyoid muscles of the left side, and finally taking a direction towards the right internal jugular vein. This vein communicates with or takes its origin from the left internal jugular or subclavian vein. The spleen was large, as also the liver; its right lobe was dislocated below the umbilicus. The gall-bladder was filled with gall-stones. No other organs were much changed. The brain, however, was not examined.

*CASE 5.—Large cervical tumour of the left side of the neck, accompanied with obscure pulsation around its base.*

Ann B—, widow, æt. 49, was admitted into the Royal Free Hospital under the author's care, February 12th, 1867, with a large tumour occupying the left side of the neck. She had suffered for six months from pain in the neck and left infra-clavicular region darting through the chest; at the expiration of this time, severe cough came on, and shortly after, she observed a fulness just above the left collar-bone. Upon her admission there existed a hard, bulging, and very slightly elastic tumour with some diffused impulse at its base. At the root of the neck the swelling corresponded to the inner two thirds of the left clavicle, extending upwards and inwards; it dislocated the larynx and trachea to the right side; externally, the swelling pushed the trapezius muscles upwards and backwards. The skin was red and livid in patches, over the surface. The face, forehead, and nose were of deep leaden tint. Very great dyspnœa existed, with violent paroxysmal cough. No abnormal signs detected within the chest, but the left radial pulse was weakened.

19th.—Dyspnœa greatly increased, amounting to veritable orthopnœa; cough almost incessant and exhaustive, accompanied by scanty mucous expectoration; severe pain in the head. The tumour presents the appearance of a large carbuncle. As no intra-thoracic change is obvious beyond



consonating râle, the great dyspnœa and cough are probably caused, for the most part, by the mechanical pressure of the swelling. Still, the existence of the pain in the chest for six months, leaves the diagnosis doubtful.

March 1st.—The swelling presents some points of indistinct fluctuation. Black, gangrenous patches are seen here and there over the surface, dyspnœa and cough still urgent; great constitutional distress.

20th.—A large slough separated, followed by copious purulent discharge. Immediate relief to the cough and dyspnœa; return of larynx and trachea to their normal position.

April 1st.—Nearly convalescent, though still complaining of pain below the left clavicle; the large wound filled up by granulation.

Two cases of subperiosteal abscess have been noticed; one occurred in a young woman, and was situate just above the left mamma, and connected with disease of the rib. The second case occurred in a male, and was situate on the centre of the sternum. These examples of abscess are interesting from their occupying the site of intra-thoracic aneurisms, and occasionally receiving concussion impulse from the heart. No difficulty, however, ordinarily attends their diagnosis.

*Remarks.*—Whenever a pulsating tumour which involves the root of the neck to any extent is first submitted to observation, and is unaccompanied by a sufficiently accurate previous history, there is, generally, a felt difficulty of diagnosis, whatever the actual position of the tumour may be. Such difficulty may occasionally arise from an inability to determine the real nature of the tumour, but most frequently (an aneurismal character being assumed) it consists in isolating the vessel engaged.

The general rules laid down for guidance in this latter particular are based almost mainly upon the position of the tumour, and the relative state of the circulation in the

corresponding arteries of the arms and neck ; the exceptions, however, to such rules are sufficiently frequent to lessen their value to a very considerable extent.

It is not within the scope of this communication to attempt more than a brief illustration of some of these exceptions, principally from cases of aortic aneurism giving rise to secondary sacculations ascending the neck, at the ordinary site of innominate or carotid aneurism, and causing modifications in the circulation and sensibility of the right upper extremity.

Plate VI, fig. 1, represents the case of a man, an Irish drover, admitted into the Hospital under the writer's care, for (supposed) subclavian aneurism.

He had suffered for some time from pain and stiffness in the neck, right shoulder, and clavicle. Eventually a tumour *first appeared above the right clavicle near its sternal end*, and gradually ascended the neck, as seen in the drawing. There was an entire absence of pulse in the right radial, ulnar, and brachial arteries. The carotid artery was apparently felt beating above the sac. The left radial pulse was of normal volume, but quickened in pulsation, ranging at 90. The motility of the right arm was decidedly lessened. This was a case of aneurism of the ascending aorta, and although the innominate artery was involved, it was a part of the aortic sac which mounted up the neck.

Plate VI, figs. 2, 3, represents a second example of aortic aneurism sending loculi up the neck in the site of innominate aneurism. One of these tumours mounts at the episternal notch, and a second, just at the outer border of the right sterno-mastoid muscle. In this case, James P—, the man, suffered from violent pain in the shoulder and right arm, with subsequent impairment of sensation and motion in this extremity ; marked atrophy of the arm followed. There was considerable weakening of the circulation at the wrist.

The difficulties that attend the differential diagnosis of innominate aneurism proper are in tolerably direct pro-

portion to the part of the trunk affected. When the origin of the vessel is alone involved (so far as the author's experience extends), it is *invariably* combined with aneurism, either true or false, of the ascending aorta. This form of innominate aneurism thus loses all individuality (so to speak), and merges into the general pathology of aortic aneurism. It has absolutely no clinical history of its own. If, as at times (though with extraordinary rarity), an aneurism should limit itself to the mid-portion of the trunk and anterior wall, it may remain for a time, or even throughout its entire course, intra-thoracic, descending and moulding itself upon the aorta, evoking all the physical phenomena and symptoms of aneurism of the ascending and transverse aorta, or it may additionally, by one or more loculi, ascend the neck either on the tracheal or acromial side of the sterno-cleido-mastoid muscle. So that should a pulsating tumour, with or without cervical extensions, occupy the space corresponding to the second and third costal cartilages of the right side, and upper portion of the sternum, and present the ordinary symptoms of aneurism, though we may be practically safe in locating the disease in the aorta, it is not from any inherent difference of signs of symptoms, but from the rarity alluded to, of intra-thoracic innominate aneurism.

It is only when the distal end of the trunk becomes the seat of aneurism that accuracy of diagnosis is attainable, though, in such a case, the origins of the subclavian and carotid arteries often share the dilatation. Certain signs and symptoms have been ascribed to innominate aneurism. Their value is high, directly as intra-thoracic disease can be eliminated; it becomes, as has been just shown, extremely small as the evidence for such disease increases. This is by far the most important point in diagnosis. The only possible source of error is the rare fact of an aortic sac arising from the convex wall of the arch, and mounting up the neck. Valuable aid is afforded by the knowledge of some antecedent local injury, or violent and unusual effort (for very many of such cases are traumatic). These points

determined, should severe pain of the right shoulder, clavicle, right side of the heart, neck, and arm, with local venous congestion or œdema precede the appearance of a pulsating tumour which, emerging from beneath the right sterno-clavicular articulation, and often displacing the head of the clavicle during its ascent, distends the space between the heads of the sterno-mastoid muscle, and fills the *episternal notch*—should there be a marked weakening of pulsation in the right radial and carotid arteries, pressure upon one or other artery lessening the impulse on the tumour—should a murmur, loudest over the tumour and at the sterno-clavicular articulation, diminish downwards, yet ascend the arteries on the right side, if pervious, such murmur being absent upon the left side, we may with tolerable safety diagnose an aneurism of the terminal portion of the innominate artery; at least such were the rules which led to a successful diagnosis in Case 1.

## DESCRIPTION OF PLATES IV, V. AND VI

### PLATE IV.

*Fig. 1, Case 1. John V.— Case of traumatic aneurism of the innominate artery, June 12, 1865.*

*Fig. 2, ditto, August 27. Tumour greatly diffused towards right shoulder and left sternum-mastoid muscle.*

*Fig. 3, ditto, September 1. Great extension of swelling round the neck (outline).*

*Fig. 4, ditto, September 5.—Tumour more diffused; ecchymosis of skin, &c (outline).*

*Fig. 5, ditto, September 8. Condition at moment of death.*

### PLATE V.

*Fig. 1, Case 2. M. A. E. Spontaneous true aneurism of the ascending and transverse aorta.*

*Fig. 2, Case 4. Deborah W. Vast aneurism of the aorta, with secondary sacculation above sternal end of left clavicle. November 20th, 1866*

*Fig. 3, ditto. Ditto, February 2nd, 1866.*

### PLATE VI.

*Fig. 1. Case of aneurism of the ascending aorta, involving the innominate artery in an Irish drover.*

*Fig. 2. Second case of aortic aneurism sending loculi up the neck in the site of innominate aneurism. Case of James P—, July 3rd, 1866.*

*Fig. 3. Ditto, ditto, August 4th, 1866.*



*Fig. 1.*



*Fig. 2.*



*Fig. 5.*



*Fig. 4.*



*Fig. 3.*





Fig. 1.



Fig. 2.



Fig. 3.





Plate IV.



*Fig. 1.*



*Fig. 2.*



*Fig. 5.*



*Fig. 4.*

*Fig. 3.*



plate V.



Fig 3

W. West del.



Fig 2



Fig 1

Engraved by J. H. Ford del.

1. The first part of the document is a list of names and addresses of the members of the committee.

ON THE REPAIR  
OF  
ARTERIES AND VEINS AFTER INJURY.

BY

HENRY LEE, F.R.C.S.,  
SURGEON TO ST. GEORGE'S HOSPITAL,

AND

LIONEL S. BEALE, F.R.S., F.R.C.P.,  
PHYSICIAN TO KING'S COLLEGE HOSPITAL, AND PROFESSOR OF PHYSIOLOGY  
AND OF GENERAL AND MORBID ANATOMY IN KING'S COLLEGE.

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Received May 28th.—Read June 25th, 1867.

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FROM numerous experiments made by Dr. Jones<sup>1</sup> he came to the conclusion that when an artery was divided, three concula were formed in the process of repair—one of blood externally, one of lymph just within the extremity of the canal, and one of blood within its cavity. "I have called that of lymph a *conglulum*," he observes, "because it may be considered a distinct substance—effused in such a quantity as to form a mass of a determinate figure." When a ligature is applied to an artery in the usual way the wounded internal surface of the canal, Dr. Jones says,<sup>2</sup> being kept in close contact by the ligature, adheres. "It seems to be entirely owing to the effusion of lymph that this union is effected."

<sup>1</sup> Jones on 'Hemorrhage,' p. 63.

<sup>2</sup> *Ibid.*, p. 160.

Hunter in the same way believed that the lining membrane of the blood-vessels is subject to inflammation like other points, and that inflammation may terminate there as elsewhere in adhesion and suppuration. He says, "In cases where I have had the opportunity of inspecting veins after death, I have found the inflammation in the adhesive state; in some places the sides of the vein were adhering, and in others the inner surface of the veins was furred over with coagulable lymph."<sup>1</sup> From the account given of the first case in which Hunter tied the femoral artery for popliteal aneurism, in the 'London Medical Journal' for 1786, it is evident that his object was to procure adhesion of the opposed sides of the vessel. For this purpose he employed four ligatures, as appears from Sir E. Home's account of the operation (p. 295). Having, he says, disengaged the artery from its lateral connections with the knife, a double ligature was passed behind it by means of an eyed probe, and the artery tied by both portions of the ligature, but so lightly as only to compress its sides together. A similar application of ligature was made a little lower down. The reason for passing four ligatures was to compress such a length of artery as might make up for the want of tightness. On the ninth day after the operation there was considerable discharge of blood from the part where the ligatures passed out, and a tourniquet was applied. On the 8th of March, the operation having taken place in the previous December, the wound broke out again. The patient was ultimately dismissed as cured on the 8th of July following. It is evident from this description that Hunter expected adhesion of the opposed sides of the artery to take place, and as evident that such adhesion did not occur. What Hunter meant by adhesion he himself tells us: "In adhesive inflammation the vessels, being enlarged, begin to separate some portion of coagulating lymph with some serum, and also red globules, and throw it out on the internal surface, probably through the exhaling vessels, or perhaps open new ones, and cover

<sup>1</sup> 'Transactions of the Society for the Improvement of Medical and Chirurgical Knowledge,' vol. i, p. 23.



*Fig. 1.*



*Fig. 2.*



*Fig. 3.*



EXPERIMENT II.—A donkey having been provided by Mr. Field, veterinary surgeon, an acupressure needle was left in contact with the main artery above the knee for three whole days. The animal was then killed. The artery, as shown by the accompanying specimen,<sup>1</sup> was red, and its coats apparently injected; but there was no trace of any effused lymph upon its lining membrane.

A patient in St. George's Hospital had the femoral artery tied for an aneurismal tumour. The patient died at the expiration of thirteen days, secondary hæmorrhage having recurred several times before the patient's death. The artery was tied with a silver wire which had very nearly eaten its way through the lining membrane of the distal portion of the artery. The interior of the vessel at this point is represented in the accompanying drawing by Dr. Westmacott (see Plate VII). The indented outline of the lining membrane can be distinctly traced, and may be seen to be quite free from any plastic effusion upon its surface. The tube is here partially closed by a decolorised coagulum, which has the slightest possible adhesion to the lining membrane of the artery; small portions of the same material adhere not very closely to the lining membrane near its termination.

Again, in Pirrie's work on 'Acupressure,' a case is related in which after an amputation of the thigh bleeding was commanded by acupressure needles under, and loops of wire over the vessels (Case V). The patient died thirty-six hours afterwards. The superficial femoral artery and vein were both included in the loop, and before the vessels were opened it could be distinctly seen that the artery contained a coagulum extending upwards from the needle for an inch and three quarters, and that the vein was empty to nearly the same extent. The artery and vein were both free from any trace of inflammation, and their coats were perfectly entire and unimpaired where they had been em-

<sup>1</sup> The specimens referred to are now in the Museum of St George's Hospital.

braced by the needle and loop. The obliterating coagulum in the artery was composed of three pieces adherent to each other and very slightly to the interior of the artery. (pp. 72, 73.)

That the process by means of which an artery or a vein when injured is repaired depends, as a rule, upon a different process to that which has hitherto been understood by *adhesive inflammation*, appears from the following observations :

An incision about an eighth of an inch in length was made by Mr. Lee through all the coats of the carotid artery of a horse, on October 29th, 1866 (fig. 1). The artery of the leg was also opened in two places close together, as shown in fig. 2. On November 1st, three full days after the operation, the animal was killed, and the vessels were carefully removed and examined. Upon slitting up the vessel with scissors a clot was discovered which extended from the opening for two inches in one direction and about one inch in the other. This dark red clot could be traced into the almost colourless material which plugged up the opening which had been made in the vessel as represented in fig. 1, but in no part whatever was the clot adherent to the lining membrane. A large quantity of blood had been effused into the areolar tissue forming the external coat of the artery, and had distended its meshes enormously, forming a firm mass more than half an inch in thickness, extending around the vessel for about two thirds of its circumference, as shown in the figure. The coagulum occupying the wound in close contact with the divided elastic coat of the artery was almost colourless, but in thin sections a few red blood-corpuscles could be detected here and here. Pretty equally distributed through it and at very short intervals were bodies having all the appearance of ordinary white blood-corpuscles. This colourless fibrin-like substance, rich in white blood-corpuscles, adhered to the divided arterial coats with tolerable firmness, and it is even possible that by its contraction it may have drawn towards each other the lips of the wound. In order to examine this rapidly-formed

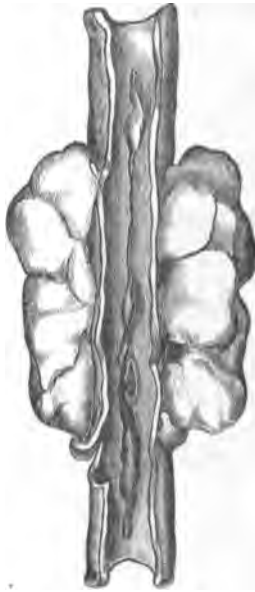
tissue minutely, in the hope of ascertaining precisely its nature and origin and the mode of its formation, that small portion of the artery where the opening had been made was removed and, after having been transferred to carmine fluid,<sup>1</sup> was hardened in glycerine containing a very little acetic acid. When in a proper state, thin sections were made so as to include the arterial coats and adventitious tissue adhering to the cut surfaces. These sections were made in a direction at right angles to the longitudinal cut, fig. 4. The fibres of the circular fibrous coat encircling the artery are therefore seen lengthwise (*b*, *c*), while the longitudinal fibres forming the innermost portion of the arterial tunic are cut across transversely or obliquely (*a*). A thin section is represented in fig. 4 under a magnifying power of twenty-nine diameters, and in fig. 5, the portion marked *e* in fig. 4 is represented magnified 700 diameters.

One of the openings in the small artery of the leg, fig. 2, with its plug projecting for about the twentieth of an inch above the inner surface, is represented about the natural size, in section, in fig. 3; and in fig. 6 a section of a part of the same is seen under a power of 40 diameters. Figure 7 shows the structure of the laminated adventitious tissue represented in fig. 6, and fig. 8 some of the amorphous matter which was continuous with this and is evidently of the same nature, magnified 700 diameters.

It is a point of the greatest interest to determine the nature of the transparent tissue shown in figs 4, 5, 6, which occupies the opening made in an artery three days after injury, for this is the material by which the wound is temporarily repaired. In it the changes which result in the complete healing of the artery, and thus bring about permanent repair, take place. After careful examination of the texture itself and consideration of various facts previously ascertained in other inquiries,—upon the formation of fibrin in the blood, the development of tissue, and the formation of lymph upon serous membranes in inflammation,—we have

<sup>1</sup> For the composition of the carmine fluid, see 'The Microscope in its Application to Practical Medicine,' 3rd edition, 1867, p. 53.

Fig. 1.



Carotid artery of a horse, three days after operation. The opening or notch a little below the middle of the vessel and passing from this is a clot which extends in both directions. Blood is extravasated in considerable quantity in the areolar coat of the vessel. Natural size.

Fig. 2.



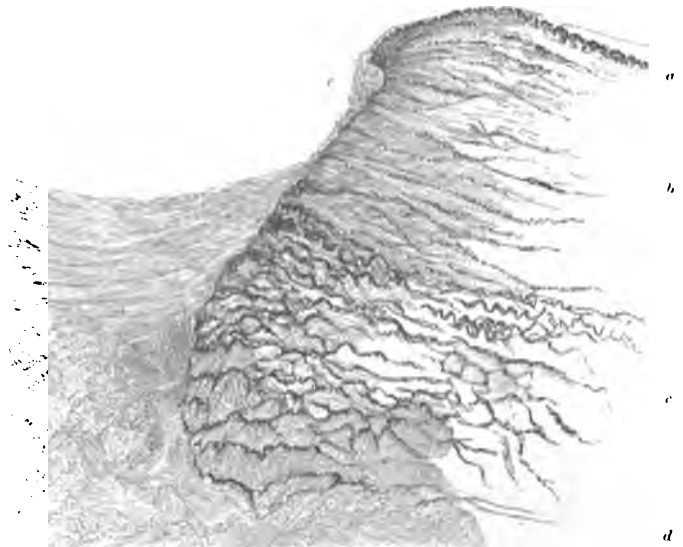
Artery, leg of a horse, in which punctures had been made, 14 days after the operation. Wound is already closed, and fibrous matter projects above internal surface, as shown in section in Fig. 3. Natural size.

Fig. 3.



Section through artery, about middle of one of the opening a, Fig. 2. Showing fibrous matter projecting above the level of the internal membrane of the artery. Natural size.

Fig. 4.



Transverse section through carotid artery at the seat of injury, showing one of the cut edges and the fibrinous material covering the wound. a, uniform longitudinal fibres cut across. b, inner portion of circular fibrinous coat of the artery. c, outer and d, inner portion of the same. d, areolar coat. On the right, the vessel is surrounded with a mass of fibrinous material which passes into the areolar fibrinous material covering the wound. x 20.



arrived at the conclusion that this adventitious tissue occupying the wound is closely allied to, though probably not identical with, ordinary fibrin and is deposited from the blood, or rather is formed through the agency of the living germinal matter of the blood—the so-called white blood-corpuscles and corpuscles which have been detached from these.<sup>1</sup> Although there is a difference in the microscopical characters of ordinary fibrin deposited from the blood, and undoubted lymph formed upon the surface of a recent wound or upon a serous membrane in inflammation, we think this difference may be due to the different conditions under which the deposition of the insoluble material occurs. At any rate we have seen fibrin undoubtedly deposited from blood which exhibited no fibrous or “fibroid” appearance whatever—fibrin which exhibited delicate lines or striæ like lymph—and fibrin which could scarcely be distinguished from certain specimens of true fibrous tissue in process of development. And on the other hand we have seen specimens of unquestionable lymph recently formed upon the surface of the peritoneum which could not, we think, have been distinguished by its microscopical characters or its behaviour with acetic acid or potash from true fibrin.

Many facts we have observed lead us to the conclusion that the formation of what is called fibrin, lymph, and fibrous or connective tissue, is due to the conditions present and the rate at which the changes proceed, rather than to a difference in the properties or powers of the living matter which produces these substances. And we believe that the so-called colourless corpuscles of the blood, lymph, and chyle, and the corpuscles resulting from their division and subdivision may give rise to fibrin, to lymph, or to fibrous tissue. In the formation of lymph the small portions of living germinal matter known as white corpuscles or bodies derived from these which have escaped from the blood through the slight fissures in the walls of the over-stretched capillary vessels, are the active agents. In all

<sup>1</sup> “On the Germinal Matter of the Blood; with Remarks upon the Formation of Fibrin,” by Lionel S. Beale, ‘Trans. Mic. Society,’ Dec., 1863.

organizable exudations such masses of living germinal matter exist, and to these must be attributed the production of the material giving to the exudation its peculiar characters. If the substance be poured out upon the surface of a serous membrane, the germinal matter in it will live and grow, and continue to form a kind of fibrous tissue probably in consequence of being very close to the blood-vessels and receiving a supply of nutrient material. If on the other hand the fluid holding in suspension masses of germinal matter be poured out upon the surface of a mucous membrane, reaching it by passing first into some of the numerous glands or follicles opening upon the surface, the thick layer of epithelium which separates it from the blood-vessels will interfere with the free supply of nutrient material. The germinal matter will die, and the mass can never become organized. In this case, although its origin and nature are the same, it is designated as *non-organizable* in contradistinction to that from the former which is called *organizable*. Whether it dies and is thrown off as foreign matter or lives and becomes an integral part of the body organism depends upon its position and its relation to the blood, not upon any peculiarities in the exudation itself. We cannot admit that all inflammation exudation masses represent a fluid holding *no germinal matter* but *only cells*. Small portions of germinal matter are constantly suspended in it, and although ultimately overpowered, these constitute the only active matter in the exudation.

When we have observed this kind of discharge taking place in the foot, which has a circulation in it, the areolar tissue in the neighbourhood of the vessels seem to show that in the disintegration and removal of its excess elements, the white blood-corpuscles increase and multiply and are in no probability a local formation.

In fig. 11 some of the fat-cells in the areolar coat of a vein, with multitudes of little granular cells in the intervals, are represented. The preparation from which this drawing was taken was obtained from the outer part of the ear three days after it had been opened by Mr. Lee. Already the colouring matter in the extravasated blood



Fig. 5. Part of wound marked *e* in Fig. 4, magnified 500 diameters. The fibrinous matter extends over the inner surface of the artery, and in various situations white blood corpuscles are seen embedded in it. The masses of germinal matter (nuclei) of the inner part of the arterial tube are well seen. They are little larger, close to the seat of injury, than in other parts of the vessel.

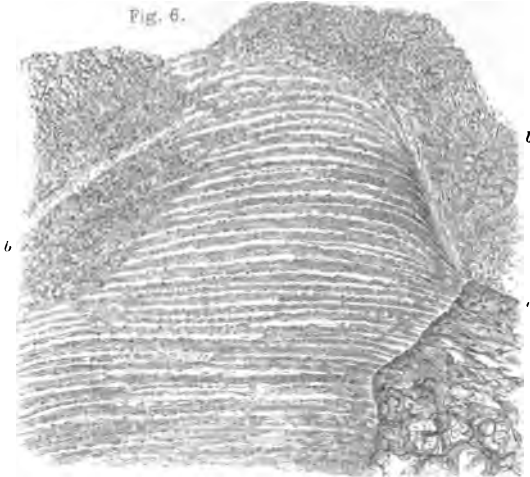


Fig. 6. Part of wound marked *b* in Fig. 4, magnified 500 diameters. The fibrinous matter extends over the inner surface of the artery, and in various situations white blood corpuscles are seen embedded in it. The masses of germinal matter (nuclei) of the inner part of the arterial tube are well seen. They are little larger, close to the seat of injury, than in other parts of the vessel.

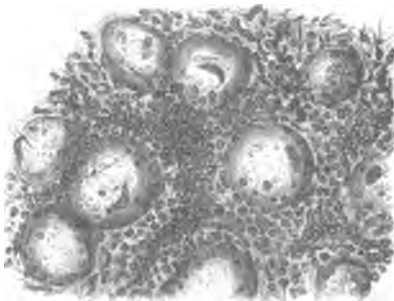


Fig. 7. Part of wound marked *b* in Fig. 4, magnified 500 diameters. The fibrinous matter extends over the inner surface of the artery, and in various situations white blood corpuscles are seen embedded in it. The masses of germinal matter (nuclei) of the inner part of the arterial tube are well seen. They are little larger, close to the seat of injury, than in other parts of the vessel.

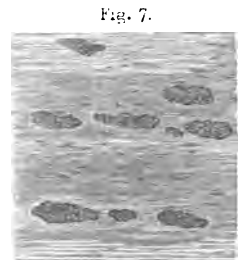


Fig. 8. Laminated fibrinous material, from Fig. 6, showing masses of germinal matter (white blood corpuscles), now coagulated, and fibrin-like substance or formed material.  $\times 500$ .

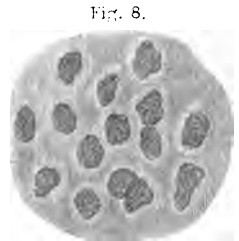


Fig. 9. Fibrinous matter not exhibiting laminar structure, showing germinal matter (white blood corpuscles), uncoagulated and fibrin-like substance formed by them.  $\times 500$ .



Fig. 10. Vein showing old cavity, probably resulting from bleeding.



Fig. 11. Vein with opening, three days after operation.



organizable exudations such masses of living germinal matter exist, and to these must be attributed the production of the material giving to the exudation its peculiar characters. If the substance be poured out upon the surface of a serous membrane, the germinal matter in it will live and grow, and continue to form a kind of fibrous tissue probably in consequence of being very close to the blood-vessels and receiving a supply of nutrient material. If on the other hand the fluid holding in suspension masses of germinal matter be poured out upon the surface of a mucous membrane, reaching it by passing first into some of the numerous glands or follicles opening upon the surface, the thick layer of epithelium which separates it from the blood-vessels will interfere with the free supply of nutrient pabulum. The germinal matter will die, and the mass can never become organized. In this case, although its origin and nature are the same, it is denominated an *unorganizable exudation* to distinguish it from the former, which is called *organizable*. Whether it dies and is thrown off (unorganizable), or lives and becomes an integral part of the body (organizable), depends upon its position and its relation to the blood, not upon any peculiarities in the exudation itself. We cannot admit that an inflammatory exudation consists merely of a fluid holding *in solution soluble matters* only. Small particles of germinal matter are invariably suspended in it, and although hitherto overlooked, these constitute the only active matter in the exudation.

Moreover, our observations upon the changes taking place in the blood which has extravasated into the areolar tissue in the external coat of the vessels seem to show, that in the disintegration and removal of its lifeless elements, the white blood-corpuscles increase and multiply, and are in all probability actively concerned.

In fig. 11 some of the fat-cells in the areolar coat of a vein, with multitudes of little granular cells in the intervals, are represented. The preparation from which this drawing was taken was obtained from the outer part of the vein three days after it had been opened by Mr. Lee. Already the colouring matter of the extravasated blood

has in great part disappeared, and the fatty matter of the fat-cells is in many cases tinged by it of a deep-brown colour. The little bodies, like white blood-corpuscles so very numerous, were evidently multiplying rapidly when the specimen was removed from the body of the animal. Here and there the remains of red blood-corpuscles could be discerned pale and indistinct. That the corpuscles figured in the tracing consisted of living germinal matter, and were not altered red blood-corpuscles, was evident. For, first, they were readily stained by carmine fluid; secondly, they varied much in size and were evidently multiplying; and lastly they were spherical, exhibiting nuclei in their interior and had that delicately granular appearance so characteristic of the white blood-corpuscle, pus-corpuscle, and other bodies composed of living germinal matter. It seems to us most improbable that these masses of germinal matter had been derived from the connective tissue corpuscles; for, first, the latter were not found to be larger than in the normal state or to exhibit any appearance of proliferation, nor were the bodies in question more numerous near the connective tissue corpuscles than in other situations. Secondly, they were so very numerous that it appears to us most improbable that they could have resulted from proliferation of the connective tissue corpuscles in the time. On the other hand, their general characters, their number, and the mode of their distribution, are such as we should expect to find if they were the descendants of white blood-corpuscles which had escaped from the vessels with the blood, and being freely supplied with nutrient pabulum in consequence of their proximity to capillary vessels, had grown and multiplied.

We now pass on to consider more particularly how the material closing the opening in the wounded artery was produced. The facts we have observed seem to us to prove that it was formed from the blood flowing in the vessel. It appears to us that it could not have resulted from changes in an exudation poured out by the vasa vasorum, for the quantity of coagulated blood in the external coat of the artery must have so compressed these vessels as to prevent

circulation through them. And even if blood flowed slowly through them it is not easy to understand by what means the exudation could have passed through the very thick coagulum, and have reached the lips of the wound. Neither is it reasonable to infer that the lymph-like substance resulted from an exudation poured out by the elastic coats of the artery itself, for in the first place, it must be remarked that the elastic tissue of an artery is not a texture which yields coagulable lymph: secondly, the lymph-like substance covered for some distance from the wound the lining membrane of the artery which was uninjured as well as the cut surfaces; and thirdly, the material in question passed gradually into decided blood-clot. Nor are we disposed to regard this as a substance formed by the nuclei or masses of germinal matter of the elastic fibres or of the muscular fibres of the arterial tunic (although these are much more numerous than represented by anatomists), for in the specimens in question we found them only slightly larger in the neighbourhood of the injury than in other parts of the artery, while we know from other observations that where new growth is the result of the increase and division and subdivision of the germinal matter of the normal texture, vast collections of germinal matter can be traced to these bodies. In fig. 5, some of the portions of germinal matter of the artery are seen to project from the divided coats, but the corpuscles in the lymph are separated from these, and there is nothing to lead us to infer that the latter have in any way descended from, or originated in, the masses of germinal matter or nuclei of the arterial tunic.<sup>1</sup> On the other hand the corpuscles in question exactly resemble those found in ordinary fibrin, and they are scattered pretty evenly through the new tissue just as we should expect if they had been derived from the blood. In figs. 7 and 8 some of these bodies with the formed material (lymph or fibrin) are seen magnified 700 diameters.

With the help of the facts we have ascertained we shall

<sup>1</sup> Unfortunately the engraver has not rendered this drawing as well as could be wished.

now attempt to describe in the order of their occurrence, the phenomena which we think occur during the first three or four days after an opening has been made in an artery in cases where the injury is repaired. If the free escape of blood from a small wound made through all the coats of an artery be prevented by closure of the external wound, clots are of course formed in the areolar tissue and among the other textures external to the vessel, and thus the escape of blood gradually ceases. In our specimens the blood in the areolar coat of the artery was found to extend for some distance from the seat of the injury.

The opening *in the elastic coat of the vessel* is not, however, immediately occupied by blood, but, as has been shown, is gradually filled up with a perfectly colourless substance. This material, which resembles the fibrin found in some aneurismal sacs, is, like that substance, deposited from the blood layer after layer until the space is filled up. Sometimes, as in the artery represented in figs. 2, 3, and 6, the process continues until an actual elevation, projecting above the level of the inner surface of the artery, is formed.

The thin laminæ of transparent fibrin are well seen in figs. 4 and 6, and from their arrangement it is evident that they have been formed from the blood which flowed along the vessel, and not from any material poured out from beneath by the vasa vasorum or from the arterial tissues. We do not, however, regard the material as a mere deposit of fibrin from the blood, but are disposed to think that it is formed by the agency of the white blood-corpuscles, which we know would adhere to the surface of the blood-clot which occupies the lower part of the wound. It seems probable that these masses of germinal matter, as they slowly move over the surface, form the material allied to fibrin figured in the drawings.<sup>1</sup> This substance, it must be remembered, has been produced within three days,

<sup>1</sup> With reference to these supposed movements see a paper "On Contractility as distinguished from purely vital movements," by Dr. Beale, 'Mic. Journal,' July, 1864. Also 'The Microscope in its Application to Practical Medicine,' 3rd edition, 1867, p. 63.

and yet it exhibits a certain definiteness of arrangement. It adheres imperfectly to the surface of the artery, but firmly to the comparatively rough lips of the wound, and by its slow contraction it no doubt draws these towards each other. Thus we believe is formed, layer after layer, a temporary tissue, which, backed up as it is by a thick clot, is strong enough to resist the ordinary lateral pressure of the blood.

The subsequent changes taking place in this fibrin-like material which result in the permanent closure of the wounded artery have not yet been investigated. We are, however, convinced that by the method of research we have adopted, it will be possible to determine whether the new fibres which enter into the formation of the new permanent fibrous tissues, result from the masses of germinal matter (white blood-corpuscles) of the temporary adventitious tissue described by us, or from those (nuclei) belonging to the normal texture of the artery which may gradually increase in number near the cut surface of the vessel, and thus extend from this point into the temporary texture occupying the wound. There is, however, no doubt that masses of germinal matter which are the direct descendants of white blood-corpuscles do possess remarkable formative power, and observations made some time since by Dr. Beale render it almost certain that white fibrous tissue and epithelium may be formed by these. There are reasons for thinking that yellow elastic tissue may likewise, under certain conditions, result, while it is certain that the higher tissues, such as organic muscle and nerve, never originate from these bodies. Into this highly interesting and important part of the inquiry we hope to enter on another occasion.

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#### DESCRIPTION OF PLATE VII.

The femoral artery tied for an aneurismal tumour. W. P—, æt 46. Operation, March 21st, 1867; death, April 3rd, 1867.

A. Distal portion.

B. Portion nearest the heart.





ON A CASE  
OF  
MUSCULAR ATROPHY,  
WITH  
DISEASE OF THE SPINAL CORD AND MEDULLA  
OBLONGATA.

BY  
J. LOCKHART CLARKE, F.R.S., &c.,  
AND  
J. HUGHLINGS JACKSON, M.D.,  
ASSISTANT-PHYSICIAN TO THE LONDON HOSPITAL, AND PHYSICIAN TO THE  
NATIONAL HOSPITAL FOR PARALYSIS AND EPILEPSY.

COMMUNICATED BY  
SAMUEL SOLLY, F.R.S., PRESIDENT.

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Received May 28th — Read June 26th, 1867.

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"Mrs. H—, æt. 38, was always delicate. Her father died æt. 62, neither of apoplexy nor paralysis. Always subject to menorrhagia. On the death of her husband she was compelled to work very hard. In June, 1864, she fell down stairs and hurt her right hand, and especially the right thumb. The hand ever after felt weak, but she continued her business as needlewoman until the commencement of 1865, when she had severe menorrhagia. She then began to complain of severe pain in the right arm, neck, and shoulder, and found that she could hardly hold a needle or lift the arm to her head. There was little or no pain in the hand and



forearm; but she complained that they felt generally much colder than on the left side. At this time all the muscles of the hand and forearm were much wasted. She took tinct. of nux vomica, and tinct. ferri sesquichlor., and in two months her health was greatly improved; she had less pain, and could hold a needle and work, and her hand did not appear so wasted or cold. She now married, became pregnant, and continued well for three months, complaining of very little pain in the shoulder. In September, 1865, her husband died suddenly. She was much shocked and never felt well after. Her face began to waste and lose expression; she complained of pain in the neck; weakness of the left hand and arm, as well as the right, and her legs dragged after her. Her voice changed; she did not pronounce words as usual; she never complained of any loss of sensation, except that her limbs sometimes felt cold and 'asleep.' As pregnancy advanced her feet became very œdematous; the pain in the neck increased; and she began to lose the power of elevating or moving her head. Her chin generally rested on the sternum, or the cheek fell on the right shoulder; and she was unable to move her head from either position, except by bending backward or sideways the trunk. She was able to shuffle about the house, but could not dress herself. She had almost completely lost the power of moving the left arm; over the right she had some power, but not much. Her deglutition now became difficult; she often saw double. Her taste was almost gone. She was confined of a healthy child in March, 1866, and suffered great privation and anxiety. After her confinement she suffered great pain in the right arm and shoulder; she frequently squinted and had double vision."<sup>1</sup>

#### CLINICAL OBSERVATIONS.

By Dr. HUGHLINGS JACKSON.

Mary H—, æt. 38, admitted into the London Hospital under my care, April 3rd, 1866.

<sup>1</sup> The above are notes furnished by Dr. Conolly (of Aldersgate Street), who first saw the patient.

May 1st.—She lies in bed (spring-bed), and has done so ever since she came in. She cannot feed herself; she can, however, do so a little when her left hand, resting on her chest, is near her mouth. As she remarks, the chief loss of power in that arm is from the shoulder to the elbow.

*Right arm.*—It is perfectly motionless, and wasted from the shoulder to the fingers. There is no stiffness nor rigidity anywhere the fingers being quite supple; they are generally extended. There does not appear to be an absence of fat, as the fingers are rather plump. I can feel nothing at any part of the arm or shoulder which gives me any idea of muscle, except at the origin of the flexor muscles of the fingers, and here there is a feeling of something more distinct than fat under the skin. The arm generally seems bone covered by skin and fat. The forearm is very flat. The anterior fold of the axilla seems to be much wasted, but still there is apparently a little muscle. No action under the Faradaic current could be developed in any of the muscles of this limb or shoulder.

*Left arm.*—She can grasp a little with the hand, and can nearly extend the fingers, but the motions are slow and feeble. The ball of the thumb is flattened, and all the thumb muscles are wasted, and the motions of the thumb are slow and feeble. The forearm is flat in its lower two thirds. The supinators are wasted. Although she cannot lift the forearm, she can wriggle it along her chest, where it usually lies, towards her mouth, rotating it and flexing it as if making a walk with her fingers. There is muscular tissue on the upper arm, both front and back, in fair quantity. The interossei of the hand acted a little to the current, the extensors feebly.

*Neck.*—The right sterno-mastoid is lax, and if it can act it acts very little. It seems to have more tendon than the left. When the nurse raises the patient the head falls according to the patient's position, and the only motion is that she can bend the head laterally—not rotate it. The sterno-mastoids I find are of no real use in fixing or moving the head when the patient is held up.

tremulous, and it does not seem to tremble as a whole, but in waves of tremulousness. Her power of swallowing is very imperfect, and she can only take sop, mincemeat, &c. Her talking is nearly unintelligible; to me it is generally quite unintelligible, but the nurse can usually manage to make out what the patient says.

Her legs were like sticks, but the right was the one more wasted. On this side there was action, and that but slight, of the peronei and gastrocnemii only. On the left leg there was considerable action of the muscles.

The left thigh was thinner than the right, and the muscles of this side acted less than those of the right. The gluteal muscles of both sides acted considerably.

The patient lay in bed till her death, and her condition did not alter appreciably from that above described. She died July 18th, 1866.

#### EXAMINATION OF THE BRAIN AND SPINAL CORD.<sup>1</sup>

By MR. LOCKHART CLARKE.

The vessels of the pia mater on the surface of the brain were somewhat congested. The grey substance of the convolutions was dark and almost purple. In the white substance the puncta were very numerous. There was no accumulation of fluid in the lateral ventricles, but the right optic thalamus was narrow—narrower than the left, and a cyst about the size of a small pea was attached to the choroid plexus of the left side.

The medulla oblongata was below the average size. It was not, however, softer than usual, nor was it anywhere damaged by *disintegration* of tissue; but many of its nerve-cells, particularly about the floor of the fourth ventricle and calamus scriptorius, were in different stages of *degeneration*. They were not very much reduced in size, but they had lost their sharp outlines; their surfaces were uneven, and many of them

<sup>1</sup> It may be well to state that the following descriptions were written and the drawings made before I had seen the notes of the case.

contained an unusually large number of yellow or brown pigment granules, as in the degenerations of old age.

The spinal cord was soft at different parts, but particularly in the cervical and the middle of the dorsal region. Opposite the second and third cervical nerves, although the cord retained nearly its usual consistence, the anterior grey substance, on each side, was atrophied to a certain extent; for the cornua were unnaturally pointed, and the nerve-cells within them were wasted to a remarkable degree, as one of the accompanying preparations will show. Scarcely a trace could be discovered of anything resembling the large cells that belong to these parts, which were now filled instead with a multitude of small granular masses and minute stellate bodies, like some of those found in the connective tissue of the white columns.

In the middle of the cervical enlargement the right lateral column was raised into a convex swelling about half an inch in length. On examining transverse sections of the cord at this part, both the white and grey substances were found to be extensively and variously damaged. Plate VIII, fig. 1, represents exactly the right lateral half of one of these sections. *a* is the anterior white column; *b* the lateral white column as far back as the posterior lateral fissure *b'*. The caput cornu posterioris is between *a* and *a'*, fringed by the pale gelatinous substance, which is traversed and divided into areas of different shapes and sizes by bundles of the posterior roots. *c* is the anterior cornu, and *d* is the transverse commissure. Along the right or lateral border of the grey substance are numerous dark masses *b b'*, of different shapes and signs, and inclosed in a network of fibres and blood-vessels. These are the cut-ends of longitudinal bundles of fibres traversing the part which I have named the *tractus intermedio-lateralis*, and which I have shown to be connected with the lowest roots of the spinal accessory nerve. Below them, and on their left, along the border and central part of the grey substance, are several blank irregular areas, which differ considerably in shape and size, and frequently coalesce at different points, or on different sides.

These are the morbid spaces, where the grey substance had first softened and then passed through the stages of transparent granular disintegration.<sup>1</sup> By inspecting the preparation from which this figure was drawn it will be seen that these spaces are variously transparent, and rendered more or less granular by particles of disintegrated tissue. Some of them are crossed, or only partially traversed in different directions by fine nerve-fibres, or by fibres of connective tissue which had hitherto escaped destruction. In the central part of the anterior cornu is the tortuous portion of a blood-vessel, surrounded on one side by one of these morbid spaces. But not only by disintegration was the grey substance injured. Atrophy, or shrinking of particular parts, was very conspicuous in this region of the cord. The nerve-cells, especially of the anterior cornu, were wasted to a remarkable degree, as may be seen at *c*, fig. 5, and in the preparation from which it was drawn under a magnifying power of 250 diameters. When they are compared with cells taken from the same part of a healthy cord, and magnified to the same extent, as represented at *a*, fig. 5, this degree of wasting is very strikingly shown. But they are not only reduced in size, they have lost their sharpness or their regularity of outline, and all traces of their nuclei.

Nor were the lesions of structure, in this case, limited to the grey substance; for the swollen lateral column (*b*) was very much softened, and in several places was completely destroyed by patches of transparent disintegration, as represented at *e, f, g, h*, fig. 1. Between and around these areas the tissue was damaged, to a less extent, by the same kind of lesion, and was densely crowded with compound granular corpuscles of various shape and size, as represented by the dark dots in fig. 1. In the same section the posterior column on each side was considerably softened, but not otherwise altered.

Immediately below this softened swelling of the right lateral column the whole of the grey substance of the same

<sup>1</sup> For a description of this process see my paper "On Tetanus" (appendix), 'Medico-Chirurgical Transactions,' vol. xlvii, 1865, p. 264.

side was much wasted and much altered in shape, as may be seen in fig. 2. The caput cornu posterioris was reduced to about half its ordinary size, at the extremity of a long slender neck; while a large portion of the *anterior* cornu, corresponding to *i* of the *left* side, had entirely disappeared. A little lower down the shape of the *right* lateral half of the grey substance was nearly normal, but the anterior cornu (*c*, fig. 3) was considerably reduced in size. At the lower third of the dorsal region, the antero-lateral column of the *left* side was raised into a softened swelling of considerable size. The grey substance, also, was much altered in shape; it had lost its sharpness of outline, and in many places was drawn out in streaks which were blended or mixed up with the softened white column. For some distance lower down the entire thickness of the cord was much firmer and much more healthy; but opposite the eleventh and twelfth dorsal nerves the deep strata of the posterior columns were soft, and the antero-lateral grey substance was much reduced in size, as shown in fig. 4 (*c*, *l*). Here we find that the large group of cells (*k*, fig. 4) constituting the posterior vesicular column has entirely disappeared on the *left* side. Nearer the middle of the lumbar enlargement the cord was again much firmer, but the anterior cornu on each side was smaller than natural, and the cells which they contained had degenerated to a certain extent, although they were not very much reduced in size. Through the lower third of the lumbar enlargement both the white and grey substances were rather softer, but retained their natural shape and general appearance. Indeed, the whole of that portion of the cord which supplied nerves to the lower extremities was very much less damaged than that which gives origin to the nerves of the upper extremities. The conus medullaris, or tapering extremity of the cord, was perfectly healthy.

## DESCRIPTION OF PLATE VIII.

Fig. 1.—Right lateral half of a transverse section of the spinal cord.

- A. The anterior white column.
- B. The lateral white column.
- B'. The posterior lateral fissure.
- C. The anterior cornu.
- D. The transverse commissure.
- a, a'. The caput cornu posterioris.
- b, b'. Dark masses enclosed in a network of fibres and blood-vessels, along the right border of the grey substance.
- e, f, g, h. Patches of transparent disintegration in the lateral column.

Fig. 2.—

- a, a'.
- i. Left anterior cornu.

Fig. 3.—c. The anterior cornu.

Fig. 4.—c, l. Antero-lateral grey substance considerably reduced in size.  
k. Group of cells constituting the posterior vesicular column.

Fig. 5.—c. Wasted nerve-cells of the anterior cornu, &c.  
d. Ditto, magnified.







ON A CASE  
OF  
CONCUSSION-LESION,  
WITH  
EXTENSIVE SECONDARY DEGENERATIONS OF  
THE SPINAL CORD,  
FOLLOWED BY GENERAL MUSCULAR ATROPHY.

BY  
H. CHARLTON BASTIAN, M.A., M.D. LOND.,  
LECTURER ON PATHOLOGICAL ANATOMY, AND ASSISTANT-PHYSICIAN  
TO ST. MARY'S HOSPITAL.

COMMUNICATED BY  
H. HAYNES WALTON, F.R.C.S.

Received June 11th.—Read June 25th, 1867.

THE following case presents many distinct points of interest. In the first place, because from a concussion not more severe than might occasionally be experienced in a Railway accident, the most unmistakeable, and even extensive, lesions of the spinal cord were produced at the time, and recognised after the patient's death. Then, though these primary and other extensive secondary lesions of the spinal cord were recognisable with the greatest ease after the organ had been immersed for a period in chromic acid, yet at the time of the autopsy itself, when the organ was in the fresh condition, no morbid appearances were detected even by careful scrutiny, and that for reasons which I shall hereafter be able fully to explain.

limb from the bed. The bladder is paralysed, and the urine ammoniacal. The bowels were moved soon after the accident, but have not been opened since. The breathing is purely diaphragmatic, the intercostal spaces falling in at each inspiration: number of respirations per minute, 32. Has a little bronchitis, and has a great difficulty in coughing up the phlegm. Pulse 88, soft. Temperature in axilla 97° Fahr. Already there is a large bed-sore over sacrum four or five inches in diameter. He complains of soreness and stiffness in the neck, and of slight pain in the neighbourhood of the first and second dorsal vertebræ; but no fracture or displacement can be detected there. (Mr. Haynes Walton writes, "he was raised in bed for me to examine his back, but there was no mark, nor any tenderness except on the upper part of the dorsal region; but this was really very slight, considerable pressure and percussion being scarcely complained of.") Ordered:—*Ol. Ricini* ʒss, *statim* (by means of which bowels were moved—he himself being quite conscious of the fact, though unable to exercise any voluntary control); full diet, with 4 oz. of port; to be placed on a water-pillow, and the bed-sore to be dressed with glycerine.

July 8th.—Has not slept well, being much troubled with startings in the legs (the first time since the accident). In other respects, in the same state as when admitted.

9th to 13th.—The startings are very troublesome—does not sleep much.

14th.—Startings chiefly confined to the right leg; is able to move the toes of the left foot a little.

17th.—A small slough has come away from the sore, granulations healthy.

21st.—Is gradually gaining a little power in the extensors of the right forearm; can move the fingers a little. The urine now dribbles away from him, even when there are but a few ounces in the bladder; complains of pain in the lower part of the abdomen and along the urethra: there has been a slight purulent discharge from the urethra for the last day or two.

24th.—Seems rather low, the voice is weak, and his speech

drawling. States that his voice is completely changed since the accident. Takes food pretty well; has 6 oz. of port wine in addition to brandy.

August 1st.—Complains of pain in right leg; nothing can be seen to account for it. There is no tenderness at any part of the spine, even when heavy pressure is made.

5th.—Hips and shoulders are a little rubbed; sore on back healthy. Is gaining a little more power over the right hand. Seems to be improving in general health, and is much more cheerful than he was. Is now propped up in bed at his own request.

18th.—Is gaining more power in his left leg; often complains that his legs pain him. Sore over sacrum much healthier.

Through a change of house-surgeons the notes unfortunately cease at this stage. Mr. Haynes Walton tells me, however, that though in the early part of August the patient could move his right arm a little, yet this very soon began to undergo a rigid contraction, which steadily increased till the wrist almost touched the shoulder. Up to this time, also, he was only ordinarily thin, but he soon began to waste perceptibly day by day, in spite of a nourishing diet with plenty of stimulants. For the last two or three months, also, he was often sick and vomited his food. The bronchial tubes became loaded with thick mucus, and at times he seemed likely to suffocate, because he had such difficulty in expectorating. For a long time he passed his urine involuntarily, but afterwards he could retain it, though he suffered great pain during its passage, and latterly blood came away with it. Bowels were never moved without the aid of castor oil. Throughout, sensation seemed to be scarcely, if at all, impaired in the paralysed or other parts of the body. From the first to the last he was on a water bed, and everything was done to prevent other bed-sores from forming, but in vain. His position was continually changed and adjusted, until he became too weak to bear the fatigue which this induced. His mind was clear till the end. He was literally dying for weeks. His appetite got less and less, whilst his

desire for stimulants increased. During the last seven weeks he took daily 2 oz. of brandy, 6 oz. of port, and two bottles of stout with two pounds of beef jelly. He died on December 31st, 1865.

*Autopsy thirty-six hours after death.*—Body emaciated to a most extreme degree; large sore over sacrum; no scar or appearance of former wound higher up in back; no irregularity of vertebral spines; thighs and knees rigidly flexed; right elbow rigidly flexed.

*Brain* presented no abnormal appearance. After removing *vertebral* arches, these appeared perfectly natural—there was no displacement or irregularity in any part. The *spinal cord* was in no way compressed. On cutting open *dura mater*, the vessels on surface of cord were seen to be large and turgid with blood. No wasting or alteration of shape was observed in any part, and on section in the upper cervical region, through middle of brachial enlargement, and in various parts of dorsal and lumbar regions, no morbid change was detected; the internal vessels were also somewhat turgid with blood, but the consistence of the organ was good, and the sections to the naked eye presented a healthy appearance. *Pericardium* dry. *Heart* healthy, small, weight 7 oz.; right cavities containing semifluid blood. No fat on surface. Both *pleuræ* dry and free from serum. *Left lung*, old adhesions about apex; deposits of tubercle and superficial puckering also in this situation; a few small granular patches of tubercle in other parts of lung; weight 14 oz. *Right lung* much more firmly adherent and more solidified about apex; also containing tubercle scattered through other parts; weight 19 oz. *Peritoneum* dry. Areolar tissue around organs also quite dry and tough, so as to cause some difficulty in removal of kidneys. *Liver* large; very pale; moulded to shape of abdomen; tissue more resistant than is ordinarily the case with fatty liver; weight 50 oz. *Spleen* healthy, somewhat small; weight 5 oz. *Kidneys*, both organs in much the same condition; large, very pale, and considerably congested. The congestion was well seen on surface, when capsules, which could be stripped off freely,

escape detection, even after a careful examination. The occurrence of cases of this kind should, however, strongly impress upon us the necessity of not passing a too hasty verdict upon organs, in which we have failed at an ordinary post-mortem examination in recognising well-marked structural changes, such as might have been expected to have been present, considering the nature of the symptoms presented during life. In this case, I examined, almost immediately after the autopsy, portions of the nervous tissue taken from various parts of the cord, with the aid of the microscope, and at once found that all parts of the organ—from the lumbar enlargement up to the medulla oblongata—contained variable quantities of well-developed granulation corpuscles, or “compound inflammatory globules,” as they were unfortunately named by Gluge. Having thus satisfied myself as to the existence of morbid changes in the organ, I cut segments of it off and put them aside to harden in a solution of chromic acid. After immersion for a certain time in this fluid, the areas of degeneration became most easily recognisable by the naked eye, on the surface of freshly-cut segments of the cord, owing to the fact that the diseased tissue, though hardened in the same way as other parts, had not become stained by the chromic acid. Thus the diseased tracts preserved their original dead white colour, whilst the adjacent healthy nerve-tissue had been stained to the usual extent, and presented a yellowish-brown hue. By this difference in colour alone, and even by a naked-eye examination, the distribution and extent of the various areas of degeneration, which I am about to describe, could be easily detected throughout the whole extent of the cord which was thus preserved. It is much to be regretted that I did not preserve the whole organ, since there is every reason to believe that some important lesions must have been situated in a part of the cord occupying the mid-cervical region, which was not kept. Not realising the full importance of preserving the whole of the organ at the time, I put aside, for subsequent examination, only the medulla oblongata attached to about one half of the upper portion of the spinal cord, the brachial enlargement, portions

other healthy fibres. By the eighth day, however, changes are much more easily recognisable; the medullary sheath has become obviously opaque, the double contour of the fibre on each side has become irregular and interrupted in places, owing to a kind of strangulation of the medullary matter; whilst by the tenth day, or even sooner, this strangulation has gone on to actual segmentation of the white substance into portions of various sizes. During the succeeding days the segmentation still progresses; the original fragments of myeline breaking up into successively smaller and smaller portions, which finally assume a more or less spherical form and fatty aspect. These are contained within the sheath of each nerve-fibre, and completely conceal the axis cylinder. After a month or six weeks the segmentation has become more complete, the medullary matter being reduced to small globules; whilst after two or three months only granulations so fine can be seen within the nerve-fibre, that they resemble "*une poussière qui remplirait la gaine conjonctive.*" At last these granulations disappear, and we arrive at the ultimate change: the sheath of Schwann collapses, and folds upon itself and upon the axis cylinder so completely, that the nerve-fibres are scarcely distinguishable as such. Having become grey also from the disappearance of the white matter, a bundle of such altered nerve-fibres under the microscope can scarcely, at first sight, be discriminated from a bundle of fibrous tissue. Dr. Waller believed that the axis cylinder disappeared altogether, and that in cases where a restoration of function took place in the cut nerve, this was due to an actual new production of nerve-fibres amongst the débris of the old. The observations of Schiff, however, and of MM. Phillipeaux and Vulpian, show that in this respect Waller was wrong. Each of these observers had recognised the existence of the axis cylinder, after an interval of more than six months, and they maintain that when the functions of a nerve so altered are restored, this is brought about not by the generation of nerve-fibres, but by the re-formation of the myeline within the shrivelled sheaths, and around the comparatively unaltered axis cylinders of the original nerve-fibres. Thus

much concerning the actual changes in the individual fibres; but Phillipeaux and Vulpian also state that in the experiments which they made upon dogs, they found the atrophied fibres of cut nerves much more difficult to separate from one another, by a teasing process, than were the healthy fibres of an uninjured nerve. This they attributed to the fact that the fine connective tissue, which normally exists in small quantity between the fasciculi of a nerve, had become hypertrophied, and consequently more tenacious.

When Waller (after having ascertained the various changes which take place in the distal portions of cut nerves) turned his attention to the effects of section of the anterior and posterior roots of the spinal nerves, he soon made known important results as to the *direction* which this degeneration takes in sensory and motor nerves respectively; and he came to the conclusion that such atrophic changes were due to the severance of the connection between portions of the nerve-fibres and their ganglionic attachment—to the interruption, in short, of some controlling nutritive power which is normally exercised over the whole length of each nerve-fibre by the nerve-cell at one of its extremities, and from which it proceeds. This influence is usually exercised in the direction of the physiological action of the fibre; thus, when the anterior roots of the spinal nerves were cut, Waller found that the fibres in the proximal portion of the cut roots retained their normal structure, whilst those in the distal portions (from the cut extremities to their peripheral distributions) underwent the changes above described. When the posterior or sensory roots of the spinal nerves were cut, on the other hand, the distal extremities in connection with the ganglia of the posterior roots preserved their healthy structure, whilst the fibres of the proximal portions underwent the atrophic change, and by this means could be traced ascending for various distances in the posterior columns of the cord, and finally losing themselves in the grey matter. From this, Waller concluded that the nerve-cells presiding over the nutrition of the motor nerve-fibres were situated in the grey matter of the cord, whilst those for the nerves of sensation



were situated in the ganglia of the posterior roots—thus showing one definite function at least for these bodies.

Other observations have tended to show that the course of secondary degenerations generally in the antero-lateral columns of the cord may be said to be downwards, whilst in the posterior columns it is just the reverse. The exception to this rule will be stated hereafter. After what has been said, it would be needless to insist upon the important aid which degenerations of this kind afford, in enabling us to determine some of the most difficult problems in connection with the anatomy of the nervous system, by facilitating our tracing the distribution of nerve-fibres through plexuses or complex organs, such as the spinal cord. This method seems second to none for the accuracy of its results, and when used experimentally it has been named by Vulpian, in honour of its discoverer, the “Wallerian method” of investigation.

The above explanations as to the effects produced by sections of nerves, sufficiently explain the nature of secondary degenerations of nerve-tissue, since the process is perfectly similar in each case; and this enables me now to say a few words concerning the history of such degenerations. Cruveilhier was the first to discover descending secondary degenerations as a result of lesions of the brain.<sup>1</sup> These he recognised in the cerebral peduncles, in the pons, and in the medulla oblongata, though he did not succeed in tracing them into the cord. To Türck, of Vienna, is due the honour of having first called attention to these lesions in the spinal cord, in an important memoir<sup>2</sup> which was presented to the Academy of Sciences of that city in 1851, that is to say, in the same year that Waller made known the most important results of his experiments on the degeneration of cut nerves. Whilst, two years later,<sup>3</sup> Türck presented to the same academy another memoir in which he analysed thirteen cases of secondary

<sup>1</sup> ‘Anatomie Pathologique,’ livraison xxxii, p. 15.

<sup>2</sup> ‘Compt. rend. de la sect. de Mathémat. et Sciences Nat. de l’Acad. des Sc. de Vienne,’ Mars, 1851.

<sup>3</sup> ‘Compt. rend. de l’Acad. des Sciences de Vienne,’ t. xi, p. 93, Juin, 1853.

degeneration of the spinal cord, and twelve others dealing with various lesions of the spinal cord itself. Notwithstanding the important nature of these communications they appear to have attracted but scant attention, since such cases as were mentioned in Blandin's great work, and in various cases reported by others. Nearly at the same time that these were coming to the notice of his researches, another case was published by Schneider Van der Noll, and by MM. Charcot and Turner. In 1867 M. Pothier wrote a memoir on secondary degenerations of the brain, and since this time various instances of such changes in the brain and spinal cord have been recorded in the *Archives médicales de la Société de Biologie à Paris* and in the *Revue de la Société Anatomique*, at the same time by MM. Charcot, Velpeau, Jorral, and Blandin. Leyden has also published a remarkable case of secondary degeneration of the spinal cord, as a result of compression from Pott's curvature of the spine; and lastly, Blandin has recently published an admirable memoir on the whole subject, in which I have been much indebted, and in which he treats of secondary degenerations of the spinal cord resulting severally from lesions of the brain, primary lesions of the cord itself, and lesions of the posterior roots of the spinal nerves. To this work I shall frequently refer. So far as I have been able to ascertain, the case which forms the subject of the present communication is the first that has been described in Great Britain or Ireland.

These secondary degenerations, which occupy a certain extent of the columns of the cord, either throughout the whole or a considerable length of the organ, develop themselves rapidly, and also simultaneously, in the different parts of the diseased tracts. This might have been imagined

\* "Exemple d'Atrophie cérébrale avec Atrophie et déformation dans un moitié du Corps" 'Compt. rend. de la Soc. de Biolog.,' 1852, p. 19).

† 'Archiv. Gén. de Méd.,' t. ii, p. 31, 1859.

‡ 'Die grade Degenerat. des hintern Rückenmarksstränge,' Berlin, 1863.

§ 'Archiv. Gén. de Méd.,' 1865, and subsequently republished in a separate form.

from what has been already said concerning the changes taking place in cut nerves, and seeing that such degenerations involve a definite series of tissue changes, it will be easily understood that the microscopical characters, and even the naked-eye appearances, of the diseased tracts vary in different stages of the retrograde process. According to Bouchard, secondary degenerations consist of and include the following changes:—1. An atheromatous alteration of the capillaries, and the formation of granulation-corpuscles in the degenerating tissue; 2. The alteration and finally the disappearance of a more or less considerable number of nerve-fibres; 3. The formation of connective tissue which substitutes itself in the place of the atrophied nerve-fibres. It will be seen further on that I accept Bouchard's account of the first two kinds of changes only with certain qualifications. It will be recognised also that the last two changes are precisely those which take place in the distal extremity of a cut nerve, if my interpretation of the nature of the second change be accepted instead of that given by Bouchard. He speaks not only of the alteration, but also of the disappearance of the affected nerve-fibres, and says, "*Je puis ajouter qu'il ne m'a jamais été permis, de retrouver les cylindres d'axe dénudés dans les cas de dégénération secondaire, et aucune observateur n'a constaté cette persistance.*" To which I can only reply, that when portions of the secondarily degenerated tissue of a spinal cord have been treated with a solution of soda, tinted with carmine, thoroughly teased with needles, and have then been finally mounted in glycerine, nothing is more easy than to demonstrate the axis cylinders of the nerves (the extremities of which have become tinted) closely enveloped by their shrivelled sheaths. The axis cylinder certainly does *not* disappear; it remains persistent in the same way as it has been demonstrated to do in the cut nerve, and the hypertrophy of the interstitial connective tissue in the degenerating columns of the cord also has its parallel in the growth of the same elements between the wasting fibres of the nerve which has been severed from its central connections. The occurrence of crowds of granulation-corpuscles in the degenerating tracts of the

spinal cord is very characteristic of this species of change ; it was to their presence that Türk principally called attention as the result of his microscopic examination ; it is owing to their presence that we are enabled to trace with comparative ease the extent and distribution of the areas of degeneration ; whilst, lastly, it is owing to the different proportions in which these are met with, in different stages of the disease, that the affected columns vary in appearance. During the first five or six months, at least, after the setting in of these changes, the granulation-corpuscles are most thickly sown through the degenerated tracts, which either retain the dead white colour of the healthy columns, or are tinted of the faintest yellowish hue. Up to this time, too, there is not the slightest shrinking—the wasting of the nerve-fibres has been exactly compensated by the formation of granule-corpuscles and the hypertrophy of the neuroglia on interstitial connective tissue. In the later stages we may get an actual shrinking or loss of substance, owing to the gradual absorption of the granulation corpuscles and the consolidation of the fibrous tissue. In proportion as such changes occur so also do we get alterations of colour : the dead white appearance is gradually supplanted by a greyish, or semi-transparent bluish grey aspect of the diseased columns.

*Microscopical examination of the cord.*—A transverse section of the hardened cord, through the upper part of the cervical enlargement (apparently corresponding with the interval between the fifth and sixth cervical nerves), showed a large rupture extending obliquely from before backwards across the grey matter of the right side, as well as a considerable shrinking in the antero-posterior direction, and a loss of symmetry of the same side of the cord (Fig. 7, Plate X). On examining this transverse section under a low power and by reflected light, it was seen that not only were the inner adjacent portions of each anterior column opaque white (as could be ascertained by the naked eye), from the presence of an innumerable quantity of granulation-corpuscles, but that bodies of the same kind were scattered more sparingly throughout other portions of the white matter of the cord,

and also in the situation of the grey matter on the right side. On the left side, the outline of the grey matter was quite distinct, and its section presented a normal appearance; whilst on the right side, on account of the large rupture extending completely through the grey matter, together with the opacity of the tissue from the granulation-corpuscles around, the outline of the grey matter could not be detected at all. From the examination of thinner sections which were prepared by Lockhart Clarke's method,<sup>1</sup> so as to make them transparent, and were then inspected by transmitted light, it was at once ascertained that, in those regions where granulation-corpuscles had been seen most abundantly in the sections submitted to reflected light, there was a great diminution in the number of healthy nerve-fibres, and a great increase in the quantity of interstitial connective tissue. It was seen, moreover, that the edges of the ruptured nerve tissue contained a large amount of connective tissue, and that the surrounding nerve matter contained also an increased quantity of this element. From the appearance presented by the section, it seems almost certain that originally the rent in the grey matter on the right side had been most extensive, but that at the time of death a certain amount of repair had taken place, owing to the development of connective tissue, by the contractile properties of which, also, this half of the cord had been drawn in, and so had lost its symmetry.

I may as well state at once that, in the examination of this and other sections of the cord, I have always found the atrophy of nerve-fibres and the hypertrophy of connective tissue, existing in any particular part, in direct proportion to the number of granulation-corpuscles; and that in all the figures I have endeavoured to represent the relative number of granulation-corpuscles by the greater or less intensity of the dotted shading.

In a section one quarter of an inch below the last (Plate X, fig. 8) there was still a slight want of symmetry on the right side of the cord, though it was much less than in the region just above it. There was no longer any rupture to

<sup>1</sup> And also by methods of my own which will be described in the third number (November, 1867) of the 'Journal of Anatomy and Physiology' (Cambridge).

be seen in the same direction as in the last figure, but a smaller degree of direction almost at right angles to it, also extending through the grey matter, and continued outwards and forwards almost to the surface with a band of scarified fibrous tissue. The degeneration of the inner parts of the anterior columns was now about equal in amount on each side, and more uniform in degree, reaching over different parts of their surface. Two rounded and very dark patches of degeneration were seen posterior to the rupture, and somewhat lighter ones in different parts of the periphery of this half of the cord. Over other portions of it granulation-corpuscles were scattered more sparingly, and the outline of the grey matter in this half still could not be detected. The number of granulation-corpuscles in the left half of the cord was somewhat less than in the last section. The grey matter on this side still presented a healthy appearance. In sections of the cord one quarter of an inch below the last (Plate X, fig. 9) the symmetry of its two sides was almost perfect, and the outline of the grey matter could now be detected in its right half, although more than one half of its surface was occupied by a large and almost square patch of dead white tissue which was made up of granulation-corpuscles, thickly sewn amongst interlacing bands of new fibrous tissue. And in transparent sections, there could be seen, in several parts of the circumference of this patch, the sheaths of blood-vessels filled with amorphous granules of blood-pigment, of a dark olive-yellow colour (Plate XI, fig. 21), whose presence clearly indicated an original rupture of blood-vessels in this situation. The degeneration of the anterior columns is now seen most distinctly to be broader, and to present a more regular border on the right than on the left side. A considerable amount of opacity from granulation-corpuscles is seen in the white matter surrounding the right half of the grey substance, and also over nearly the whole of this half of the cord. The right half, on the contrary, with the exception of the anterior column, is seen to be freer from granulation-corpuscles than it was in the last section; a few only being seen in the inner and posterior part of the lateral column.

In sections one third of an inch below the last (Plate X, fig. 10), the opaque patch in the grey matter of the right side no longer existed; almost the whole of this half of the cord was considerably clouded, and the outline of the grey matter could scarcely be detected, whilst running through its entire extent in an antero-posterior direction was another irregular rupture across the grey matter. In the anterior part of the grey matter of the left side there was a shorter and much smaller rupture, with a slight opacity round it, and in the whole of this half of the cord, more especially around the posterior grey cornu, there was a sparing distribution of granulation-corpuscles. The anterior columns were in much the same condition as in the last section. In sections one third of an inch lower down (Plate X, fig. 11) the rupture through the grey substance on the right side no longer existed; this had resumed its healthy appearance. In the centre of the grey matter of the left side, however, two small solutions of continuity were seen. The anterior columns presented much the same appearance as before, but the remainder of the right half of the section was much clearer than it is represented in the last figure—the principal area of degeneration being a somewhat semicircular one in the posterior part of the lateral column. A few corpuscles were seen also, on this side, in the part of the posterior column next the grey matter, and in the lateral column in front of what Clarke calls the *tractus intermedio lateralis*. A few granulation-corpuscles were also scattered over the left half of the section, but they were very sparse, and only formed a distinct aggregation in the posterior part of the lateral columns, in a situation corresponding with the much larger and more marked area of degeneration in the opposite half of the cord. In sections made two thirds of an inch below the last, and just below the cervical enlargement (Plate X, fig. 12), no ruptures are seen in any part; the large and well-marked areas of degeneration in the anterior columns are somewhat different in shape, the peripheral portions being broader and more extended towards the lateral columns, whilst that on the right still continues rather more extensive than the one on the left side. There

It is noteworthy that the altered granules in the anterior part of the right lateral column, whilst in the posterior portion there is a continuation of the area seen in the last section, which is of the same wedge-like shape, with its apex at the anterior extremity of the cord, and its base, which becomes granular, just anterior, lying close to the periphery of the cord, as in the anterior part of healthy tissue in its outer side. In the lower dorsal region, in the upper half of the cord, there are some scattered granular corpuscles as in the last section, but the grey matter and other parts of the white substance present a healthy appearance. This arrangement continues up to the thoracic, and was found to prevail with but slight variation in the mid-dorsal region (Plate X, fig. 13). Whilst we note a continuation of the alteration in shape of the areas of degeneration in the anterior columns, such as we saw commencing in the last section. At the periphery these patches now extend fairly as far as the lateral columns. The scattered granules in the anterior part of the right lateral column have disappeared, whilst the area in its posterior part has again become more semicircular, and its outer boundary almost reaches the periphery in this region. The few scattered granules in the corresponding area of the opposite side exist as in the last section, whilst the remaining parts have a healthy appearance.

In the lower part of dorsal region, one inch above the lumbar enlargement (Plate X, fig. 14), there was an even more appreciable alteration in the shape of the anterior areas of degeneration, their outline having become more like what it was in the lower cervical region (Plate X, fig. 10), owing to the resumption of the bluntly-rounded shape of the posterior or central extremities of these areas, which had become acutely wedge-shaped in the mid-dorsal region and contiguous parts, and also to the greatly diminished extent of the peripheral expansion of these areas. In the outer part of each, not far from its external extremity, there is a distinct notch, owing to the encroachment of healthy fibres on the diseased areas. The patch in the posterior part of the right lateral column is very distinct still, and now undoubtedly extends



quite to the surface of the cord. The scattered corpuscles in the corresponding region of opposite side are rather fewer in number, but are also situated rather more externally than they were in the sections above. The gradual wearing out of the diseased fibres and the diminution in the number of granulation-corpuscles were well seen in sections lower down, made through various parts of the lumbar enlargement, and have been represented in Plate XI, fig. 15, showing a section through the upper part of lumbar enlargement, and one inch below the last; in fig. 16, which represents a section made three eighths of an inch lower still; and in fig. 17, which was copied from a section made through the lower third of the lumbar enlargement, three fourths of an inch from the commencement of the *filum terminale*. In these it will be seen that the disease disappears first, most notably from the outer parts of the patches in the anterior columns, although at the same time a thinning-out of the diseased fibres takes place also in the inner parts of the same areas of degeneration. In the posterior part of the lateral columns, also, the atrophied fibres and granulation-corpuscles are gradually replaced by healthy nerve-fibres.

From this description it will be seen that the principal one of the original lesions or ruptures of the cord was situated in the upper part of the cervical enlargement, though there is every reason to believe that one or two other important lesions must have been situated in the portion of cord immediately above this, which was unfortunately not preserved. The large oblique rupture represented in Plate X, fig. 7, gradually diminished in extent, and one quarter of an inch lower down became continuous with another smaller rupture in an opposite direction (Plate X, fig. 8). Opposite this first section there had been the greatest damage to the cord, which had been followed by a certain amount of shrinking on the injured side. From the fact that already degeneration was well marked in the inner part of each anterior column, it is almost certain that there must have been another lesion higher up, which had severed the connection between these nerve-fibres and their ganglionic cells. It will be seen,



of half an inch. A very small rupture also existed in the grey matter of the opposite side. On each side of the posterior half of the rupture on the right side there were considerable patches of degenerated tissue, the inner being in the outer part of the posterior columns (which I shall speak of hereafter), whilst the outer was in the posterior part of the lateral column. Seeing that a well-defined area of degeneration is to be recognised in all the remaining sections in this same posterior part of the lateral column, and that this place corresponds with the situation occupied by the fibres of that portion of the left anterior pyramid which decussates, it would seem almost certain that this area of degeneration is due for the most part to a solution of continuity of these very fibres which have been well named, collectively, by Bouchard, the "*faisceau encéphalique croisé ou externe*." It would seem, also, almost certain, since no such area is to be recognised in Plate X, figs. 7, 8, or 9, that this band of fibres must have been torn across by the extensive rupture shown in fig. 10, and that in this situation (somewhere about the middle of the cervical enlargement) these fibres must have been situated fairly in the grey matter of the right side, and have been just about to emerge into the posterior part of the lateral column. Thus we get most valuable anatomical evidence as to the place of emergence from the grey matter of this bundle of fibres. In the lower part of the cervical enlargement and in the upper dorsal region the area of degeneration does not extend to the surface of the cord; in the mid-dorsal region and thence downwards, however, the outermost fibres are situated quite at the surface of the cord.<sup>1</sup>

<sup>1</sup> This is in accordance also with the experience of Bouchard, who, towards the end of his memoir, speaking of a case in which the secondary degeneration or sclerosis of the lateral column of the cord was extremely well marked, says:—"Je dois dire que la sclérose atteignait la meninge vers le milieu de la région dorsale, au lieu de former une bandelette complètement entourée par la substance blanche saine: J'avais donc été trop absolu dans la première partie de ce travail en disant qu'aucune fibre du faisceau encéphalique croisé n'arrivait au contact de la pie-mère."

The *direct encephalic fibres* contained in the cord—in addition to the band in the posterior part of each lateral column which is formed by the descending fibres of the pyramids—are lodged in the inner part of each anterior column. This band, which Bouchard names "*faisceau encéphalique direct ou interne*," seems to be made up of those fibres of the anterior pyramids which do not decussate. This conclusion has been arrived at from the fact that in cases of extensive cerebral lesion leading to secondary degeneration of the spinal cord, the diseased tract can be traced downwards through the whole extent on the same side as the lesion: except the corresponding half of the pons, the pyramid of the same side, and a short space along the inner part of the anterior fibres of the cord on the same side, and the posterior part of the lateral column on the opposite side. From the observations made in the present case it would seem, as I have already pointed out, that this descending band only reaches the lumbar region, opposite the middle of the cervical enlargement. And even when, as in no case of secondary degeneration, according to Bouchard, has there been noted any alteration of the roots of the spinal nerves, it is presumed that the degenerated fibres do not pass into them, but that they terminate gradually at different levels by passing into the grey matter of the cord, and there being continuous with certain of the large nerve-cells of the anterior cornu. The number of fibres in this lateral band gradually diminishes in the lower parts of the cord, though certain of them may be traced, as in the present case, even into the lower part of the lumbar enlargement. But, whilst Bouchard admits this also, he says, with regard to the direct encephalic fibres in the cord, that the longest of them do not extend farther than the middle of the dorsal region. This dictum seems directly contradicted by what we have seen in the present case, where the degeneration of the inner part of the anterior columns only wears itself out most gradually in descending to the lower part of the lumbar region, probably from the gradual passage of its fibres into the grey matter. But this thinning-out of the diseased fibres is not perceptible at all until we get

to the lower dorsal region, and the diminishing areas can be traced with the greatest ease, even into the lower third of the lumber enlargement.

In addition to the bands of encephalic fibres occupying the inner part of the anterior column and the posterior part of the lateral, each antero-lateral column is made up of a number of *commissural* fibres which belong entirely to the spinal cord, and which also have their nutritive centres at their upper extremities. These occupy the whole intermediate space between the two bands of encephalic fibres, and their office seems to be that of longitudinal commissures between groups of cells in the grey matter, situated slightly above one another. Bouchard names them "short commissural fibres;" and he speaks of certain others, much fewer in number, and situated just in front and to the outer side of the external encephalic band (also having their nutritive centres above), which extend for a much greater distance through the cord. These he names "long commissural fibres."<sup>1</sup> Concerning the latter class of fibres, the present case cannot be said to furnish any evidence either positive or negative; but many of the scattered granulation-corpuscles in figs. 7-11 (Plate X), doubtless mark the situation of atrophied short commissural fibres, which, at a level just below the cervical enlargement, have all disappeared with the exception of a few in the anterior part of the right lateral column (fig. 12, Plate X): in the next section represented these also have vanished.

In this, as in other cases of secondary degeneration of the spinal cord, no atrophy either of the anterior or of the posterior nerve roots could be detected.

We now come to the consideration of the degenerations of the posterior columns in the upper cervical part of the cord, and in the medulla oblongata. These belong to another category; they are all ascending degenerations. None of the original lesions which lead to these exist in the portions of the cord about to be examined. These must have been situated in that portion of the cord (below what is about to be described and above the lesion already described in the upper part of the

<sup>1</sup> Loc. cit., p. 570.

cervical enlargement) which was, most unfortunately, not preserved. Inasmuch as nothing can be said, therefore, with regard to the origins of these degenerations, all I can do will be to describe their situations as they ascend through the upper part of the cord and the medulla oblongata. They occupy the posterior columns principally, but also exist in the outer and posterior part of each lateral column.

It has been known for some time that in the most external part of the posterior portion of the lateral columns above the middle of the dorsal region, there are a certain number of fibres whose nutritive cells are situated in the grey matter of the cord at their lower extremities, and which consequently degenerate in an ascending direction. Some of these fibres have been traced upwards through the corpora restiformia into the cerebellum. Areas of degeneration due to the implication of such fibres may be seen in figs. 6-1, Plate IX, marked *c*, *c'*. They are seen to extend very far forward on the right side of fig. 6. They diminish rather gradually upwards, and an inspection of figs. 2 and 1 show that they occupy the outer and anterior part of each restiform body, and are situated just behind the dentate nuclei of the olivary bodies. The precise collocation of the fibres seems to vary somewhat as they ascend, judging from the different shapes presented by the same tract of degeneration at different levels.

In the posterior columns of the cord, secondary degenerations always take an ascending direction. These columns seem to be made up of a mixture of fibres, not distinctly separated from one another, part of which, as in the anterolateral columns, are commissural, whilst the others are continuations of the posterior roots of the spinal nerves. Of these last, some travel but a very short distance amidst the other fibres of the posterior columns and then throw themselves into the grey matter, whilst the remainder travel for long distances in the posterior columns before losing themselves in the same substance. Although these ascending degenerations were pointed out by Türck, our knowledge concerning them is still very indefinite. Concerning the different kinds of fibres in the posterior columns, Bouchard says:—"On

peut démontrer cette proposition en comparant la forme de la dégénération ascendante dans les cas de compression des racines, et de compression de la moelle elle-même. Quand la dégénération succède à une lésion des racines, elle est circonscrites sur les coupes par une portion d'ellipse, la convexité de la courbe étant au avant, et ses deux extrémités reposant sur la face postérieure de la moelle; le tissu extérieur à cette ligne est parfaitement sain. Quand il y a compression de la moelle elle-même ses fibres radicales ascendantes sont atteintes sur un point de leur parcours et vont se dégénérer au-dessus du point comprimé; cependant la figure que présente la dégénération, sur les coupes de la moelle, n'est pas la même. Au lieu d'un segment d'ellipse, on a un triangle dont la base est sur la face postérieure de la moelle, le sommet vers la commissure. C'est que la dégénération porte également sur d'autres fibres qui ont leur centre trophique à leur extrémité inférieure dans la substance grise de la moelle. Ce sont des fibres médullaires propres, comme celles que nous avons indiquées dans les cordons antero-latéraux."<sup>1</sup>

In the present case, as before mentioned, I can say nothing with regard to the precise nature of the lesions leading to the ascending degenerations of the posterior columns. This is the more to be regretted, as they are so remarkably circumscribed and symmetrical. Sections through the lowest part preserved of the portion of spinal cord presenting these lesions, displayed what has been represented in fig. 6, Plate IX. This section is from the upper third of the cervical region of the cord, exactly  $2\frac{1}{4}$  inches from the point of the fourth ventricle. Four areas are seen, constituting two almost perfectly symmetrical pairs, one of which is situated in each posterior column. The most internal patches (*a*, *a'*) have an elongated elliptical form, whose more rounded anterior extremities occupy contiguous portions of the tissue skirting the posterior median fissure in its middle third, whilst their sharply-pointed posterior extremities, almost reaching the surface, slightly diverge from one another. The outer patch of each pair (*b*, *b'*) reaches forwards as far as the grey

<sup>1</sup> Loc. cit., p. 574.

commissure, and occupies most of the anterior third of the boundaries of the posterior median fissure. From this situation each patch extends backwards and slightly outwards, closely skirting the internal areas and terminating somewhat short of the surface. Of these the area on the right side is somewhat the larger, and terminates in a small process almost at right angles, the two together looking not unlike a man's leg and foot. Following up, first of all, the areas *a*, *a'*, through sections of the lower half of the medulla oblongata (figs. 5, 4, 3, Plate IX), we see that in this situation these areas occupy the greater part of the posterior median columns of the cord. They have become almost triangular also from the widening out of their posterior extremities, and they have extended forwards, on each side of the median fissure, as far as the grey commissure, and have consequently caused a lateral displacement of the outer areas, which, in the lower section, themselves occupied this situation. In figs. 4 and 3, also, it will be seen that the number of diseased fibres has most perceptibly diminished, and that more particularly in the centre of each patch, where small areas occupied by healthy fibres may be perceived. In fig. 2, which represents a section of the medulla immediately above the point of the fourth ventricle, the posterior median columns having as usual become greatly increased in size, still show a few granulation-corpuscles chiefly scattered through their most superficial portions. The diseased fibres have evidently much decreased in number in this situation, and further than this I have not traced them. In looking now to the outer pair of patches of degeneration (*b*, *b'*), we find them, in figs. 5, 4, and 3, evidently diminishing in extent and intensity; we find also that they occupy that portion of the posterior column which in the medulla oblongata goes by the name of *processus cuneatus*, and we see, by fig. 3, that they are situated immediately on the inner side and behind the grey cornua of this same fasciculus. Tracing them onwards in fig. 2, we find them pushed further away to the side, owing to the development of the posterior median columns, though still occupying the same position with regard to the above-named



grey cornua. In fig. 1 the diseased fibres are more scattered; they are now completely in the lateral region of the medulla, forming part of the *corpora restiformia*, and in these bodies they doubtless proceeded onwards, together with the atrophied fibres of the outer and posterior parts of the lateral columns (*c, c'*), into the cerebellum, though I have myself not followed them further than the situation represented in fig. 1, as the parts above were not preserved.

A small area of degeneration on the right side marked *d* may be traced through figs. 5, 4, 3 and 2, Plate IX; further than this it could not be traced, though the fibres composing it may have passed also into the right corpus restiforme, and gone to form part of those occupying the area marked *b'* in fig. 1.

As I am unable myself to say anything concerning the anatomical relationships of the fibres entering into the formation of these tracts of degeneration in the posterior columns, I cannot do better than quote from Bouchard's memoir what seems to bear directly on the subject. Speaking of the fibres which come from the lower half of the cord, he says:—"Ces fibres qui se prolongent jusqu'à la partie supérieure de la région cervicale sont toutes logées, dans l'épaisseur des faisceaux greles et des pyramides postérieures." It would seem almost certain, therefore, that many of those which form the tracts of degeneration marked *a, a'* in my figures must be of this kind. He then adds:—"Les fibres qui naissent de la moitié supérieure de la moelle ne paraissent pas se mélanger aux précédentes, de sorte que les faisceaux sensitifs du membre inférieur et ceux du membre supérieur resteraient, isolés les uns des autres, séparés par les sillons intermédiaires postérieurs. En effet, dans un cas de compression de la moelle à la partie supérieure de la région dorsale, L. Türck a vu la dégénération occuper la partie externe des cordons postérieurs. Malheureusement il n'a pas fait de coupes dans l'épaisseur de bulbe ni de la protubérance, de telle sorte que la démonstration anatomo-pathologique de la continuation d'une partie des cordons postérieurs à travers les corps restiformes fait complètement défauts." The fibres entering into

the tract marked *b, b'* in my case seem to correspond to some of those last mentioned by Bouchard, whether or not it be correct that they have their origin in the upper part of the dorsal region. In the present case, also, they have been fairly traced upwards through the lower part of the medulla, into the corpora restiformia, thus confirming, in a new way, that anatomical distribution of some of the fibres of the posterior columns which has been previously taught to exist by anatomists.

Having now described the various morbid appearances met with in the spinal cord, I shall give a somewhat more minute account of the actual histological components of the diseased patches. These are of three kinds, viz.: 1. Atrophied nerve-fibres; 2. New connective-tissue elements; 3. Granulation-corpuscles.

*Atrophied nerve-fibres* can be detected in all parts of the degenerated columns of the cord, and it seems difficult to understand how they escaped the observation of Bouchard and others. According to Bouchard, however, this has been the case. They are most easily detected after a portion of this tissue has been treated with a solution of caustic soda for a short time, then tinted with carmine and mounted in glycerine. The axis cylinder exists surrounded by the shrivelled and folded hyaline sheath of Schwann, the medullary matter having all disappeared (fig. 20 d, Plate XI). The axis cylinders vary much in size, just as they do in the healthy nerves; and it is owing to the fact that more or less of their extremities become stained with the carmine that the atrophied nerve-fibres can be so readily discovered after the tissue has remained for some hours in a carmine solution. Here and there also in the midst of the diseased tissue we meet with nerve-fibres in a healthy condition (fig. 19 and fig. 20, e, Plate XI).

*New connective-tissue elements* exist in the greatest abundance in the sclerosed columns. Its various nuclei and cells may also be best seen after treatment with soda, carmine, and glycerine. This large quantity of connective tissue is derived from the hypertrophy of the neuroglia or normal ele-

ments of this kind which enter into the formation of the cord. In the healthy organ it forms a very delicate fibrous framework, in whose trabeculæ—gradually becoming finer as we approach the central grey matter of the cord—the nerve-fibres are lodged. Fig. 18, Plate XI, represents the normal appearance of a portion of one of the anterior columns of the cord, and shows the delicate nature of the reticulum of connective tissue; whilst fig. 19 shows the appearance of one of the diseased anterior columns magnified to the same extent. The right half of this figure exhibits the appearance presented by a very thin section when mounted in glycerine; the most striking feature being the number of large rounded or ovoidal granulation-corpuscles, each surrounded and enclosed by a rim of connective tissue containing an abundance of nuclei. The left half of the figure represents the appearance of a similar thin section mounted in Canada balsam after saturation with turpentine: here the granulation-corpuscles, being made up of molecular fat, have been dissolved, and the fibrous alveolæ in which they were lodged are fully displayed. The two methods of investigation are, therefore, most valuable. Here and there in the midst of the diseased tissue a healthy nerve-fibre may be seen. Such fibres, either having escaped rupture by the original lesion, or having issued from the grey matter below its level, still remain entire, and in connection with their nutritive cells. When portions of the diseased tracts after tinting with carmine have been teased with needles, and are then submitted to a high magnifying power, the connective tissue is found to be made up of the finest and most delicate fibres, closely beset with more or less spherical nuclei varying in size from that of a small granular speck up to  $\frac{1}{1000}$ " diameter. Some of these elements have been represented in fig. 20, Plate XI, where in addition to the fine fibres which seem to have dot-like nuclei attached to them at intervals, other somewhat coarser fibres are seen in connection with more faintly tinted, flatter cells (*c, c, c*); these vary in size and shape, and seem to be connected also (as at *c'*) with the finer fibres and dot-like nuclei. These cells contain a few rather large granules in their interior, but no distinct

nucleus. That marked *c* strongly resembles a multipolar nerve-cell, with the exception that it contains no nucleus. There can be no question, however, that it is precisely similar in nature to the others marked *c*, *c*, *c*, and situated as they all were in the midst of the anterior white columns of the cord, it seems only possible for us to look upon them as connective-tissue elements. The large nuclei were apparently unconnected with fibres, and all intermediate sizes could be traced between them and the small dot-like forms. They existed in the greatest abundance, and seemed to represent only different ages of one and the same element. All alike became deeply stained with carmine, whilst the other description of cells of which I have spoken were only tinted of a delicate rose colour.

*Granulation-corpuscles* seem always destined to be misunderstood. They were formerly brought prominently forward by Gluge, who looked upon them as the products of an inflammatory process: and it was in great part owing to the prevalent reception of this doctrine that ordinary softening of the brain, in which such corpuses invariably occur, have been so long and erroneously regarded as necessarily inflammatory in nature. The real facts are, however, as Virchow and other pathologists have lately insisted, that such elements may be met with in any place where cells exist which are gradually losing their vitality. Almost all tissues that are falling into decay, therefore, whether physiological or pathological, may exhibit such structural elements; and whether we meet with them in the brain or spinal cord, in the lungs from degeneration of its epithelium, in the kidney from changes in the same element, or in cancerous or other cellular tumours—in all cases we may safely assume that they are formed from cells whose vitality is gone, or fast going—from cells which are gradually undergoing a process of retrograde fatty metamorphosis, preliminary to a complete molecular disintegration. I think there is no evidence whatever in proof of the assumption that such bodies originate by the gradual aggregation of molecules originally separate, and I should scarcely have considered it necessary to make these observations now had not Bouchard, in his valuable memoir,

assigned two modes of origin to the granulation-corpuscles which are so abundant in secondary degeneration of the spinal cord, both of which are, I believe, alike untenable. He seems to think that, as a rule, they are formed by the aggregation of fat-granules resulting from the molecular disintegration of the myeline of the nerve-tubes; though he suggests that some of them may also result from "la transformation granulo-graisseuse de gouttes de myéline."<sup>1</sup> Not to speak of the undoubted process by which bodies of this kind originate in other organs, and therefore the probability that they are produced by a similar process in this, we consider that a microscopical examination of the elements in question sufficiently disproves the theories of Bouchard, inasmuch as around most of the smaller granulation-corpuscles a very thin envelope may be detected—the original cell-wall greatly distended; whilst after tinting with carmine we can distinguish with the greatest ease, in the interior of each, and more or less covered with granules, a large spherical or ovoid nucleus, very similar to those which are found so abundantly in the free condition amidst the connective tissue fibres. This appearance I have represented in fig. 20, *a* (Plate XI), and from it I think we are almost bound to conclude that these bodies result from the fatty degeneration and repletion of nucleated cells. From what precise cells they originate, however, does not seem certain, and in the cord which I have examined the process of their formation could not be traced. It represented too late a stage of the degeneration, and in it these corpuscles were all fully formed. I should fancy from the character of the contained nucleus that the cell must have been developed around one of the original free spherical nuclei, which, perhaps, soon began to undergo a process of fatty degeneration. This, however, must be left a matter of doubt for the present.

*Vessels.*—In this as in other species of degeneration of nerve-tissue, the capillaries and small vessels assume what has been called an atheromatous appearance. The actual change, however, is not one of atheroma, neither is it

<sup>1</sup> Loc. cit., p. 234.

attained in the walls of the vessels at all. The appearance results from an accumulation of fatty elements around the vessel, though within its so-called lymphatic sheath. This Bowerbank also imagined to be the real condition, although he did not feel quite sure upon the subject. I, however, ascertained by an examination of some of the vessels taken from the cord in its most contracted state that they were closely enveloped and not merely surrounded by fatty regenerated nuclei.<sup>1</sup> The fatty degeneration is originally produced by a proliferation of these along the inner surface of the perivascular sheath, and when in a state of fatty regeneration they almost exactly resemble some kinds of small pus-corpuscles. Whilst these vessels are in their increased abundance within the sheaths of the vessels, many of the nuclei which also exist in the outer substance of the lymphatic sheath are much enlarged, and filled with fat-particles.

The nerve-fibres in the unimpured grey matter throughout the tract presented a normal appearance. In no part of its extent were interrupted or pigmentarily degenerated nerve-cells met with.

In estimating the probable course and sequence of the changes met with in this degeneration it would seem to be as follows: the primary or initial changes commence in the nerve-fibres, and the others are more or less direct consequences of these. Thus, when a number of nerve-fibres have been severed from their nutritive ganglionic centres, and the process of degeneration of the white matter which has been described is in progress (whilst no obstruction or mechanical impediment to the flux of blood through the part exists), it seems evident that a redistribution of the nutritive pabulum amongst the elements of the tissues becomes inevitable. For, the nerve-fibres which form such a large part of the bulk of

<sup>1</sup> *Loc. cit.*, p. 287.

<sup>2</sup> It seems to me almost certain that many of the smaller areas of granular degeneration, which have been described by Lockhart Clarke in his examinations of the cord in cases of tetanus and other morbid states, are in reality produced by this same kind of distension of the lymphatic sheath around the vessels.

the tissue, are no longer in a condition of nutritive activity, they do not select fresh material from the blood, and consequently a much larger share of pabulum is at the disposal of the intervening connective tissue. Being supplied with an excess of nutritive fluid, and, as the nerve-fibres atrophy, having more space at its disposal, it is not to be wondered at that hypertrophy of the connective tissue follows. The lymphatic sheaths of the blood-vessels have also an increase of nutritive fluid at their disposal, and this may be the first stimulus leading to the increased proliferation from their lining nuclei which has been spoken of. Their subsequent fatty degeneration, and also that of some of the newly formed connective tissue-cells, leading to the production of granulation-corpuscles, may be partly due to an instability of constitution in the elements themselves (owing to their rapid and irregular formation), and partly, as suggested by Bouchard, owing to the great abundance around them of the products of the retrograde metamorphosis of the nerve-tubes, which may still further disorder their nutrition. That these causes may be instrumental in bringing about the fatty change seems probable, from the fact that after a time the loaded condition of the vascular sheaths gradually diminishes. Granulation-corpuscles also after a time seem no longer to be produced; though when once formed of course they take some time to disappear, as this can only be brought about by molecular disintegration and absorption. These bodies are, therefore, to be found for a considerable time after the commencement of the process of degeneration, but it is now well known to those who have studied the subject that after some months the number of these bodies to be found in the diseased tracts gradually diminishes, in proportion to the length of time from their first formation; so that after the lapse of eighteen months or two years they may have all disappeared, leaving in the now shrunken tracts only atrophied nerve-fibres, and greatly hypertrophied connective tissue.

From what has been said it will be seen how nearly allied the process of cerebral or spinal softening is to that of these secondary degenerations. The regressive changes in the

Although we have no clinical records of a difference in the amount of power possessed over the two extremities, there is reason to believe from the much greater extent of the degeneration in the posterior parts of the right lateral column that the capability of voluntary movement must have been less on this side than on the left. The *startings* of the limbs—more especially of the right lower extremity—which was complained of after the first ten days and for some time subsequently, was doubtless to be attributed to the irritation of healthy nerve-fibres proceeding to the limbs, owing to the reparatory process at the seat of the original lesions in the cervical region. The secondary degenerations which must have been making progress at that time in the anterior and lateral columns, could not of themselves cause any symptoms, since the mere fact of the degeneration taking place in them was of itself evidence that they were functionally inert and cut off from their physiological and nutritive centres. The state of *rigid contraction* which the muscles of the right arm subsequently assumed, appears to have commenced about two months after the accident; beginning almost imperceptibly and gradually increasing, as it is stated to do by Bouchard.<sup>1</sup> It is certainly a question of some difficulty to ascertain the exact cause of this late rigidity coming on in paralysed parts. Some hold that it is entirely due to changes in the paralysed muscles themselves; others, such as the late Dr. Todd, ascribe to it a cerebral origin, and believe it to result from the irritation produced by the contraction and cicatrization of a brain lesion. Bouchard, however, is not a believer in either of these explanations, and thinks that in all cases, whether the paralysis be of cerebral or of spinal origin, the cause of late rigidity in the paralysed muscles is to be ascribed to changes taking place in the spinal cord itself. He believes that now we know of the invariable existence of secondary degeneration of the spinal cord in cases of apoplexy depending upon lesions of the corpora striata, optic thalami, or pons, we need no longer look for the cause of this late rigidity in the brain itself; but that it is explicable in the

<sup>1</sup> Loc. cit., p. 292.



same way as in cases of paralysis due to original lesions of the spinal cord, from a consideration of the natural progress of the secondary degenerations in this organ. He thinks, in fact, that this important symptom is due to an irritation of adjacent healthy fibres, when the new growth of connective tissue taking place in the diseased tracts, comes to press upon or encroach amongst the healthy fibres. The suggestion is ingenious and deserving of consideration, though there seem difficulties in the way of its acceptance. Time will not permit of my discussing these questions, however, and I must pass on to the next symptom—that of *pain* in the paralysed limbs. Although only slightly mentioned in the clinical record, I call attention to it now, since MM. Charcot and Cornil<sup>1</sup> have lately made some investigations as to the cause and treatment of this, which is oftentimes a most distressing symptom. They believe it to be due to a kind of hypertrophic neuritis in the affected limb, since they have found the nerves increased in volume, more vascular than natural, and having a notable increase in the thickness of their connective tissue envelopes. Whether this change is due to mere functional inertia or is more directly dependent upon the secondary degenerations of the cord is not known. It seems, however, to have an evident relation to the pains above mentioned, and, according to M. Charcot, these are not only increased by pressure along the course of the nerves, but are often soothed by the application of a blister in this situation.

The *respiration* was described as being diaphragmatic when the patient was admitted into the hospital, but it seems probable, from the condition of the cord, as well as from the length of time that the man lived without fatal engorgement of his lungs, that there was by no means complete paralysis of the thoracic and abdominal muscles of respiration. Their action was doubtless much impaired, and hence amongst other things the extreme difficulty the man had in expectorating the mucus with which his bronchial tubes were loaded for some time before his death. Fortunately, the lesions of

<sup>1</sup> 'Compt. Rend. de la Soc. de Biolog.,' 1863.

the grey matter did not extend quite so high as the origins of the phrenic nerves, and the diaphragm was therefore unaffected. Had it been otherwise—even though one side only were affected—death would, in all probability, have been very rapid.

It now only remains for me to say a few words concerning the *general muscular atrophy*, which commenced about two months after the accident, and which in the remaining four months of the man's life had reduced him almost to a skeleton. This atrophy following paralysis has been frequently noticed, and not unfrequently it has been confounded with 'progressive muscular atrophy,' which should, however, be regarded as a distinct disease. Unfortunately, having my attention so much attracted to the spinal cord at the time of the post-mortem, I altogether omitted to make any special examination of the atrophied muscles, though I did take out and put into chromic acid for subsequent examination the great semilunar ganglia of the sympathetic system. A careful inspection of them and a comparison with others removed from patients dying of different diseases, enables me to say that these ganglia were undoubtedly atrophied: they were scarcely as much as one third of their usual size, and whilst all other parts of the body were remarkable for the almost total absence of fat, on making thin sections of these bodies and then placing them under the microscope, certainly a larger proportion of thin fluid fat was seen than is usually met with in such sections. The ganglion-cells seemed to contain rather more than their usual amount of pigment; they were more highly refractive also than in other sections with which I compared them, and the nucleus and nucleolus, which are usually so apparent in these cells, could scarcely be distinguished in one out of twenty of the ganglion-cells, in sections of portions of the sympathetic belonging to our patient. These were the only abnormal conditions that I was able to detect in the semilunar ganglia, and other parts of the sympathetic system were not examined.

These changes met with in a limited part of the sympathetic system, although not very decided, will, I hope, be

sufficient to attract increased attention to the condition of the sympathetic system in other cases of paralysis followed by muscular atrophy. In addition to the muscular atrophy in this case, there were other conditions which might have been dependent upon a disease of the sympathetic system; since the post-mortem examination revealed an abnormal condition of the liver, apparently due to fatty degeneration, tubercle in the lungs, and disease of the kidneys: whilst during life there was constant vomiting. If the muscular atrophy and degeneration of viscera were in reality due to a morbid condition of the sympathetic in this case, we should have to look upon this as secondarily affected, and as a result of the primary disease of the spinal cord: whilst in progressive muscular atrophy, M. Jules Simon, a late able writer on the subject, maintains that some abnormal condition of the sympathetic system is the starting-point of the disease, which secondarily affects the muscular system and some of the anterior roots of the spinal nerves. In this view he is supported more or less entirely by Dumenil, Schneevogt, Remak, Bärwinkel, Jaccoud, and Professor Troussseau; and MM. Schneevogt,<sup>2</sup> and Jaccoud<sup>3</sup> have published most important cases in which unmistakeable fatty degeneration of the sympathetic system was met with. It is only fair to add, however, that some still adhere to Cruveilhier's doctrine of the dependence of this disease upon a disease of the anterior roots of the spinal nerves: whilst others, such as Duchenne, Virchow, and Aran believe the disease to be a primary one of the muscles themselves.

I may say in conclusion, that the prognosis does not seem quite hopeless in cases of secondary degeneration of the spinal cord, even after the supervention of late rigidity in the paralysed muscles, since M. Bouchard has seen a cure result in five such cases.<sup>4</sup> These were all cases of complete

<sup>1</sup> 'Nouv. Dict. de Méd. et de Chirurg.,' vol. iv, 1866, article "Atrophie Musculaire Progressive."

<sup>2</sup> 'Nederlan. Tijds. 'Lancet,' 1854, and Schmidt's 'Jahrb.,' 1855.

<sup>3</sup> 'Mém. de la Soc. Méd. des Hôpitaux,' November, 1864.

<sup>4</sup> *Loc. cit.* p. 297.

paraplegia, from compression of the cord due to Potts' curvature. He says: "Dans 4 cas la sensibilité et le mouvement ont reparu avec toute leur intégrité; dans 1 seul, les mouvements, sans avoir recouvré leur entière liberté, permettent cependant à la malade de marcher. Dans ce cas la paraplégie était flasque; dans les autres elle s'accompagnait de contracture." "On peut donc, en conclure que les tubes nerveux de la moelle peuvent se régénérer comme ceux des nerfs périphériques, non seulement chez l'enfant, mais encore chez l'adulte et lors même que les faisceaux dégénérés, ont été déjà le siège d'un travail d'hypergénèse des éléments nucléaires."

If what I have already stated concerning the persistence in the degenerated tracts of the axis cylinders of the nerves be borne in mind, it seems probable that the repair in these cases is brought about in the same manner as when it occurs in peripheral nerves. Here MM. Phillipeaux and Vulpian have convinced themselves that the restoration of function is due to the reproduction of myeline around the persistent axis cylinders of the nerve-fibres rather than to the production of entirely new fibres, as it was formerly imagined. Notwithstanding the evidence afforded by the recovery of the patients above mentioned, and his knowledge of the manner in which restoration of function was brought about in divided nerves, Bouchard imagined that the atrophied nerve-fibres entirely disappeared in the spinal cord; but my statement that the axis cylinders of the fibres are easily recognisable in the disease columns, though resting upon evidence indubitable to myself, receives additional confirmation from these clinical facts.

Fig 1

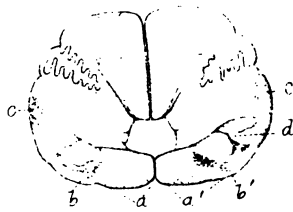
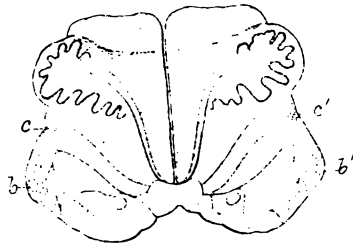


Fig 3

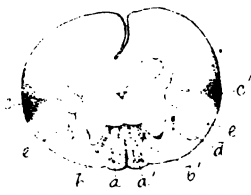


Fig 4

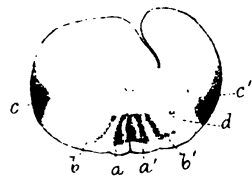


Fig 5

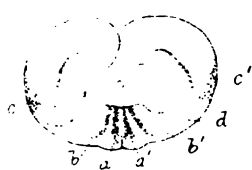


Fig 6



Fig 7.



Fig 11



Fig 8.



Fig 12



Fig 9



Fig 13



Fig 10



Fig 14.



Fig 1

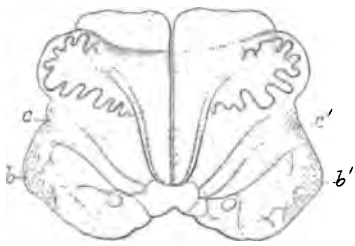


Fig 2

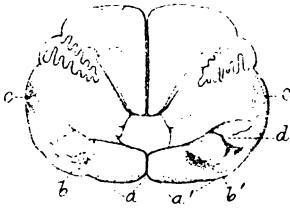


Fig 3

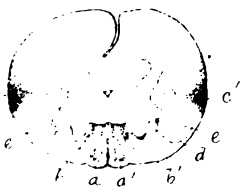


Fig 4

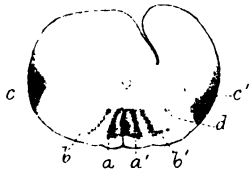


Fig 5



Fig 6



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## PLATE IX.

Fig. 6.—Transverse section through upper cervical part of spinal cord ( $2\frac{1}{2}$ " below point of fourth ventricle), showing two pairs of almost symmetrical and well-defined areas of degeneration in posterior columns of cord (*a, a'* and *b, b'*), and also unequal areas of degeneration in the lateral columns (*c, c'*). In addition there is a very slight patch of degeneration in each anterior column near the points of exit of anterior nerves.

Fig. 5.—Section  $1\frac{1}{2}$ " above last, which is on a level with the lower boundary of the medulla oblongata and of the decussation of the pyramids. Letters of reference same as in last figure, pointing to more limited areas of degeneration, with the addition of *d*, another small patch situated just beneath the neck of the right "grey tubercle of Rolando."

Fig. 4.—Section through medulla  $\frac{1}{2}$ " above last and  $\frac{7}{8}$ " below point of fourth ventricle. Letters of reference same as in last figure. The median patches of degeneration marked *a, a'* have become wider, and now contain some healthy fibres in their midst. They are seen very distinctly, by this figure and the next, to occupy the posterior median columns of the cord.

Fig. 3.—Section through medulla  $\frac{1}{3}$ " above last, and  $\frac{1}{4}$ " below the point of the fourth ventricle. Decussation of pyramids not represented in this or other figures. Letters of reference same as in Fig. 5, pointing to gradually waning areas of degeneration. The single area, *d*, on right side, is now seen to intervene between the much enlarged "grey tubercle of Rolando" and the cornu of the "processus cuneatus." The areas *a, a'* have increased in width with the posterior columns, and contain much more of healthy tissue in their midst.

Fig. 2.—Section through medulla just above point of fourth ventricle. The widened posterior median columns in this situation are almost composed of healthy tissue, and show only a very slight cloudiness externally. The areas *b, b'* and *c, c'* are more limited and much less obvious, and the same is the case with the area *d*, on the outer side of the cornu of the processus cuneatus.

Fig. 1.—Section of medulla higher up through the middle of olivary bodies. The areas *a, a'* have disappeared with the posterior median columns; the area *d* has also disappeared; whilst the areas *b, b'*, now occupying the restiform bodies, have become rather lateral than posterior; they are much fainter and more diffuse. The areas *c, c'*, in the anterior borders of the restiform bodies, have also nearly disappeared.

<sup>1</sup> Each of the sections through the medulla oblongata and the spinal cord is represented twice its natural size.

*Fig 7.*



*Fig 11.*



*Fig 8.*



*Fig 12.*



*Fig 9.*



*Fig 13.*



*Fig 10.*



*Fig 14.*



*Fig 7.*



*Fig 11.*



*Fig 8.*



*Fig 12.*



*Fig 9.*



*Fig 13.*

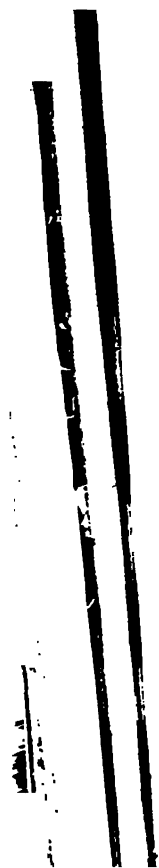


*Fig 10.*



*Fig 14.*







## PLATE XI.

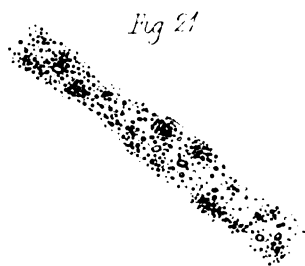
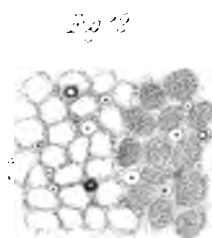
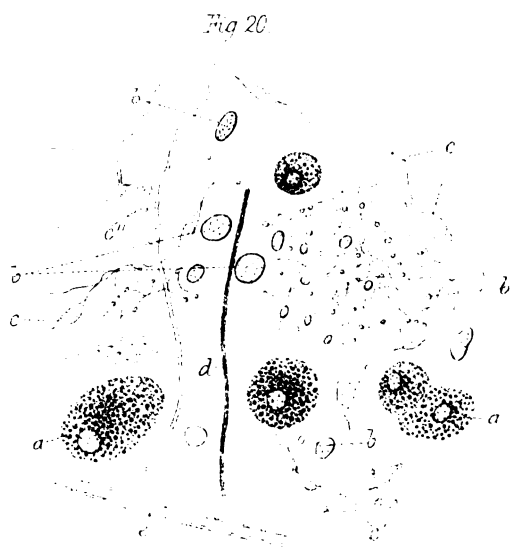
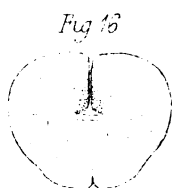
Figs. 15, 16, 17.—Sections through the upper part of the lumbar enlargement,  $\frac{3}{8}$ " below this last, and through the lower third of the lumbar enlargement, or  $\frac{3}{4}$ " from the commencement of the *filum terminale*. These show the gradual way in which the areas of degeneration terminate, and therefore the positions occupied by the longest of the degenerated fibres.

Fig. 18.—The appearance presented by a section through a healthy portion of one of the anterior columns of the cord, showing the sections of different sized nerve-fibres with intervening partitions of delicate fibrous tissue. Magnified 150 diameters.

Fig. 19.—The appearance of a section through a diseased portion of one of the diseased anterior columns. The right half of the figure gives the appearance of a thin section when mounted in glycerine, showing the abundance of large granulation-corpuscles separated from one another by richly nucleated connective tissue, whilst the left half shows the appearance of a similar section after immersion in turpentine and Canada balsam, when the granulation-corpuscles have been dissolved out and the fibrous alveoli in which they were situated are more plainly displayed. Here and there also in each half the sections of unaltered nerve-fibres are seen, and in the whole figure the sections of three blood-vessels are shown, whose walls are considerably thickened. Magnified 150 diameters.

Fig. 20.—Highly magnified representation of the different kinds of elements met with in the secondarily degenerated columns, as they appear after tinting with carmine:—*a, a, a*, granulation-corpuscles of different sizes, each having in its interior a well-marked nucleus; *b, b, b*, free spherical or ovoidal nuclei, such as exist in the greatest abundance, and of all sizes; *c, c, c*, branched cells of various shapes and sizes, which become much more faintly tinted with carmine than the preceding nuclei; *c'* shows the apparent connection between these two kinds of elements such as may be seen occasionally; *c''*, one of the largest and most extreme forms of these cells, closely resembling a nerve-cell; *d*, one of atrophied nerve-fibres consisting only of the axis cylinder and the delicate sheath of Schwann, which is closely wrapped round it; *e*, one of healthy nerve-fibres from midst of diseased tract. Magnified 400 diameters.

Fig. 21.—Appearance presented by one of the vessels just outside the large area of degeneration in the grey matter represented in Fig. 9. It is rather the sheath of the vessel which is seen loaded with amorphous blood-pigment of a dark yellowish olive colour and marking the site of a previous effusion of blood.



x 150





A THIRD AND FOURTH SERIES  
OF  
FIFTY CASES OF OVARIOTOMY,  
WITH  
REMARKS ON THE SITUATION AND LENGTH OF THE  
INCISION REQUIRED IN THIS OPERATION.

BY  
T. SPENCER WELLS, F.R.C.S.,  
SURGEON IN ORDINARY TO HER MAJESTY'S HOUSEHOLD; SURGEON  
TO THE SAMARITAN HOSPITAL, ETC.

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Received June 10th.—Read June 28th, 1867.

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My first series of 50 cases of ovariotomy is recorded in vol. xlv of the 'Transactions' of this Society, with some account of the history and progress of the operation in Great Britain. In vol. xlviii a second series of 50 cases is also recorded, with some remarks on the selection of cases for this operation. I now bring before the Society a third and fourth series of 50 cases, making in all 200 cases in which I have completed the operation of ovariotomy. I have arranged these cases in tables, as with the former series, and after a few remarks on the age and social condition of the patients, I propose to consider the situation and extent of the incision by which the operation is commenced.

If it should be acceptable to the Society, and if I should be enabled to complete another hundred operations, I may at some future time ask for attention to some observations on other steps of the operation.



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116	Dec.	21	Single	No adhesions; clamp; 10 lbs.; incision 4 inches	...	...	Died 4th day. Septic peritonitis.
117	" 1865	27	"	No adhesions; pedicle returned; 15 lbs.; incision 4 inches	...	...	Recovered.
118	Jan.	42	"	No adhesions; clamp; 28 lbs.; incision 7 inches	...	...	"
119	"	19	"	Parietal and omental adhesions; clamp; 15 lbs.; incision 6 inches	...	...	"
120	"	54	Married	Parietal adhesions; clamp; 46 lbs.; incision 7 inches	...	...	"
121	Feb.	27	Single	Parietal adhesions; clamp; 33 lbs.; incision 7 inches	...	...	"
122	"	20	"	Parietal adhesions; clamp; 28 lbs.; incision 6 inches	...	...	"
123	"	60	Married	Adhesions; clamp; 20 lbs.; incision 6 inches	...	...	Death on 5th day. Peritonitis.
124	Mar.	36	"	No adhesions; clamp; 27 lbs.; incision 8 inches	...	...	Recovered.
125	"	25	Single	No adhesions; clamp; 32 lbs.; incision 5 inches	...	...	"
126	April	31	Married	Omental adhesions; clamp and ligature; 37 lbs. solid, 8 lbs. fluid; incision 20 inches	...	...	Died, 27 hours. Exhaustion.
127	"	41	"	Adhesions; clamp; 27 lbs.; incision 5 inches	...	...	Recovered.
128	May	33	"	Adhesions; ligature; 23 lbs.; incision 8 inches	...	...	"
129	"	38	"	Adhesions; clamp; 27 lbs.; incision 5 inches	...	...	"
130	June	56	Single	Adhesions; pedicle returned; 30 lbs.; incision 6 inches	...	...	"
131	"	53	Married	Both ovaries removed; intestinal and pelvic adhesions; ligature; 23 lbs.; incision 7 inches	...	...	Died in 30 hours. Exhaustion.
132	"	34	Single	No adhesions; clamp; 60 lbs. fluid; incision 3½ inches	...	...	Recovered.
133	"	54	Married	Adhesions; pedicle returned; 33 lbs.; incision 5 inches	...	...	Died on 5th day. Septicæmia.
134	"	37	"	Both ovaries removed; parietal and intestinal adhesions; pedicle returned; incision 5 inches	...	...	Recovered.
135	July	41?	"	Omental adhesions; clamp; incision 8 inches	...	...	Died on 4th day. Peritonitis.
136	"	45	"	Adhesions; clamp; 48 lbs.; incision 8 inches	...	...	Recovered.
137	"	41	"	No adhesions; clamp; incision 5 inches	...	...	"
138	"	55	"	Adhesions; clamp; 75 lbs. fluid; incision 5 inches	...	...	Died on 9th day. Peritonitis.
139	Aug.	24	"	Adhesions; pregnant uterus; clamp on pedicle; uterine ligatures; 28 lbs.; incision 4½ inches	...	...	Recovered.
140	Oct.	46	Widow	Adhesions; clamp; incision 5 inches	...	...	"
141	"	53	Married	No adhesions; pedicle returned; 24 lbs.; incision 5 inches	...	...	Died 46 hours after. Exhaustion.
142	"	59	"	Parietal and intestinal adhesions; ligature and cautery; incision 5 in...	...	...	"

No.	Date.	Age.	Condition.	Connections; treatment of pedicle; weight of fluid or tumour; length of incision, etc.	Result.
143	1865				
144	Oct.	31	Single	Adhesions; clamp; 30 lbs.; incision 6 inches	Recovered.
145	Nov.	42	"	Adhesions; clamp; 30 lbs.; incision 5 inches	"
146	"	31	"	No adhesions; pedicle returned; incision 5 inches	"
147	"	30	"	Adhesions; pedicle returned; 28 lbs.; incision 5 inches	"
148	"	41	Married	Adhesions; clamp; incision 4 inches	"
149	Dec.	50	"	Both ovaries removed; pedicle returned; 34 lbs.; incision 5 inches	"
150	"	35	"	Adhesions; clamp and ligature; 40 lbs.; incision 5 inches	"
151	"	45	Single	No adhesions; clamp; 31 lbs.; incision 6 inches	"
152	"	41	"	No adhesions; clamp; 24 lbs.; incision 5 inches	"
153	1866				
154	Jan.	25	"	Slight adhesions; clamp; 17½ lbs.; incision 6 inches	"
155	"	20	"	Parietal and omental adhesions; clamp; 22 lbs.; incision 4 inches	"
156	"	29	Married	No adhesions; pedicle returned; 16 lbs.; incision 4 inches	"
157	"	39	"	Omental adhesions; clamp; 26 pints ascitic fluid, and 9 lbs. 6 ozs. malignant tumour; incision 8 inches	"
158	Feb.	37	Single	No adhesions; wire clamp and ligature; 52 lbs.; incision 6 inches	"
159	"	32	"	Slight adhesions; clamp; 20 lbs.; incision 6 inches	"
160	"	31	"	Parietal, omental, and intestinal adhesions; clamp; 7 lbs.; incision 4 in.	"
161	Mar.	26	"	Parietal adhesions; clamp; suppurating cyst; 8 lbs.; incision 5 inches	"
162	"	31	Single	No adhesions; clamp; ruptured colloid cyst; 30 lbs.; incision 6 inches	"
163	"	30	"	Very firm adhesions, parietal, omental, and mesenteric; pedicle returned; 24 lbs.; incision 5 inches	"
164	"	23	"	Firm and extensive adhesions; clamp; 69 lbs.; incision 4 inches	"
165	"	24	"	No adhesions; pedicle returned; 16 lbs.; incision 7 inches	"
166	April	27	"	No adhesions; clamp; 16½ lbs.; incision 7 inches	"
167	"	46	Married	Omental adhesions; clamp; 1½ lbs.; incision 8 inches	"
168	May	42	"	Omental and parietal adhesions; clamp; 26½ lbs.; incision 5 inches	"

168	May	32	Single	No adhesions; pedicle returned; 16 lbs.; incision 5 inches	...	Recovered.	
169	"	57	"	Extensive adhesions; clamp; 15 lbs.; incision 7 inches	...	"	
170	June	24	"	No adhesions; cautery; 28 lbs.; incision 4 inches	...	"	
171	July	50	Married	Slight adhesions; cautery; 23 lbs.; incision 4 inches	...	"	
172	"	25	Widow	Parietal and omental adhesion; cautery; 15 lbs.; incision 7 inches	...	"	
173	"	45	"	Parietal adhesions; cautery and ligatures; 17 lbs.; incision 7 inches	...	Died on 4th day.	Septicæmia.
174	"	28	Married	Parietal, omental, and intestinal adhesions; clamp; 28½ lbs.; incision 8 in.	...	Recovered.	
175	"	30	"	Parietal and omental adhesions; pedicle returned; 23 lbs.; incision 4 in.	...	"	
176	"	32	"	No adhesions; clamp; 13 lbs.; incision 6 inches	...	"	
177	"	39	"	No adhesions; clamp; 24½ lbs.; incision 5 inches	...	"	
178	Aug.	22	"	No adhesions; clamp; not weighed; incision 4 inches	...	"	
179	"	59	Single	Parietal and intestinal adhesions; clamp; not weighed; incision 5 in.	...	"	
180	"	42	Single	Parietal adhesions; clamp; 21 lbs.; incision 4 inches	...	"	
181	"	40	Married	No adhesions; cautery and ligatures; 15 lbs. and 18 pints ascitic fluid; incision 7 inches	...	"	
182	"	53	Widow	Slight adhesions; clamp; 28 lbs.; incision 5 inches	...	Convalescence. Death after a month.	
183	Oct.	48	Single	Omental and intestinal adhesions; clamp; 28 lbs.; incision 5 inches	...	Chronic peritonitis.	
184	"	37	Married	Omental and intestinal adhesions; clamp; 11 lbs. 13 ozs. and 7 pints ascitic fluid; incision 9 inches	...	Recovered.	
185	"	48	"	Omental adhesions; cautery; 18 lbs.; incision 7 inches	...	"	
186	"	43	"	Adhering capsule; cautery and ligature; 36 lbs.; incision 6 inches	...	Died on 5th day.	Peritonitis.
187	"	21	Single	No adhesions; cautery and ligature; 20 lbs.; incision 6 inches	...	Recovered.	
188	"	38	"	No adhesions; clamp; 14 lbs.; incision 5 inches	...	"	
189	"	32	Married	Omental adhesions; pedicle returned; 44 lbs.; incision 9 inches	...	"	
190	Nov.	37	"	No adhesions; clamp; 24 lbs.; incision 4 inches	...	Died in 42 hours.	Peritonitis.
191	"	50	"	No adhesions; clamp; 23 lbs.; incision 4 inches	...	Recovered.	
192	"	56	"	No adhesions; clamp; 14 lbs.; incision 4 inches	...	"	
193	Dec.	62	Single	Parietal and omental adhesions; clamp; 32 lbs.; incision 6 inches	...	"	
194	"	31	"	Parietal and omental adhesions; clamp and ligature; 9 lbs. 5 ozs. and 13 pints ascitic fluid; incision 10 inches	...	"	
195	"	55	"	Parietal, omental, and intestinal adhesions; clamp; 15 lbs.; incision 6 in.	...	Died in 33 hours.	Peritonitis.
						Died in 76 hours.	Septicæmia.

No.	Date.	Age.	Condition.	Connections: treatment of pedicle, weight of fluid or tumour, length of incision, etc.	Result.
143	1865 Oct.	34	Single	Adhesions; clamp; 30 lbs.; incision 6 inches	Recovered.
144	Nov.	32	"	Adhesions; clamp; 30 lbs.; incision 5 inches	"
145	"	31	"	No adhesions; pedicle returned; incision 5 inches	"
146	"	30	"	Adhesions; pedicle returned; 28 lbs.; incision 5 inches	"
147	"	41	Married	Adhesions; clamp; incision 4 inches	"
148	Dec.	50	"	Both ovaries removed; pedicle returned; 34 lbs.; incision 5 inches	"
149	"	35	"	Adhesions; clamp and ligature; 40 lbs.; incision 5 inches	Died on 22nd day. Peritonitis.
150	"	45	Single	No adhesions; clamp; 31 lbs.; incision 6 inches	Recovered.
151	"	44	"	No adhesions; clamp; 24 lbs.; incision 5 inches	"
152	1866 Jan.	25	"	Slight adhesions; clamp; 17½ lbs.; incision 6 inches	"
153	"	20	"	Parietal and omental adhesions; clamp; 22 lbs.; incision 4 inches	"
154	"	29	Married	No adhesions; pedicle returned; 16 lbs.; incision 4 inches	"
155	"	39	"	Omental adhesions; clamp; 26 pints ascitic fluid, and, 9 lbs. 6 ozs. malignant tumour; incision 8 inches	Died on 7th day.
156	Feb.	37	Single	No adhesions; wire clamp and ligature; 52 lbs.; incision 6 inches	Died on 12th day.
157	"	32	"	Slight adhesions; clamp; 20 lbs.; incision 6 inches	Recovered.
158	"	34	Married	Parietal, omental, and intestinal adhesions; clamp; 7 lbs.; incision 4 in.	"
159	Mar.	26	"	Parietal adhesions; clamp; suppurating cyst 8 lbs.; incision 5 inches	"
160	"	31	Single	No adhesion; clamp; ruptured colloid cyst; 30 lbs.; incision 6 inches	Died in 25 hours. Pyæmic fever.
161	"	30	"	Very firm adhesions, parietal, omental, and mesenteric; pedicle returned; 24 lbs. incision 5 inches	Died in 26 hours. Exhaustion.
162	"	23	"	Firm and extensive adhesions; clamp; 69 lbs.; incision 4 inches	Died in 35 hours. Peritonitis.
163	"	24	"	No adhesions; pedicle returned; 16 lbs.; incision 4 inches	Died in 52 hrs. Pulmonary embolism.
164	April	27	"	No adhesions; clamp; 16½ lbs.; incision 7 inches	Recovered.
165	"	46	Married	Omental adhesions; clamp; 16½ lbs.; incision 7 inches	"
166	May	42	"	Omental and parietal adhesions; clamp; 14 lbs.; incision 8 inches	Died on 4th day. Peritonitis.
167	"	52	"	Omental and parietal adhesions; clamp; 25½ lbs.; incision 5 inches	Recovered.

168	May	32	Single	No adhesions; pedicle returned; 16 lbs.; incision 5 inches	...	Recovered.
169	"	57	"	Extensive adhesions; clamp; 15 lbs.; incision 7 inches	...	"
170	June	24	"	No adhesions; cautery; 28 lbs.; incision 4 inches	...	"
171	July	50	Married	Slight adhesions; cautery; 23 lbs.; incision 4 inches	...	"
172	"	25	Widow	Parietal and omental adhesion; cautery; 15 lbs.; incision 7 inches	...	Died on 4th day.
173	"	45	"	Parietal adhesions; cautery and ligatures; 17 lbs.; incision 7 inches	...	Septicæmia.
174	"	28	Married	Parietal, omental, and intestinal adhesions; clamp; 28½ lbs.; incision 8 in.	...	Recovered.
175	"	30	"	Parietal and omental adhesions; pedicle returned; 23 lbs.; incision 4 in	...	"
176	"	32	"	No adhesions; clamp; 13 lbs.; incision 6 inches	...	"
177	"	39	"	No adhesions; clamp; 24½ lbs.; incision 5 inches	...	"
178	Aug.	22	"	No adhesions; clamp; not weighed; incision 4 inches	...	"
179	"	59	Single	Parietal and intestinal adhesions; clamp; not weighed; incision 5 in.	...	"
180	"	42	Single	Parietal adhesions; clamp; 21 lbs.; incision 4 inches	...	"
181	"	40	Married	No adhesions; cautery and ligatures; 15 lbs. and 18 pints ascitic fluid; incision 7 inches	...	Convalescence. Death after a month.
182	"	53	Widow	Slight adhesions; clamp; 28 lbs.; incision 5 inches	...	Chronic peritonitis.
183	Oct.	48	Single	Omental and intestinal adhesions; clamp; 28 lbs.; incision 5 inches	...	Recovered.
184	"	37	Married	Omental and intestinal adhesions; clamp; 11 lbs. 13 oza. and 7 pints ascitic fluid; incision 9 inches	...	"
185	"	48	"	Omental adhesions; cautery; 18 lbs.; incision 7 inches	...	Died on 5th day.
186	"	43	"	Adhering capsule; cautery and ligature; 36 lbs.; incision 6 inches	...	Peritonitis.
187	"	21	Single	No adhesions; cautery and ligature; 20 lbs.; incision 6 inches	...	Recovered.
188	"	28	"	No adhesions; clamp; 14 lbs.; incision 5 inches	...	"
189	"	32	Married	Omental adhesions; pedicle returned; 44 lbs.; incision 9 inches	...	Died in 42 hours.
190	Nov.	37	"	No adhesions; clamp; 24 lbs.; incision 4 inches	...	Peritonitis.
191	"	50	"	No adhesions; clamp; 23 lbs.; incision 4 inches	...	Recovered.
192	"	36	Single	No adhesions; clamp; 14 lbs.; incision 4 inches	...	"
193	Dec.	62	Married	Parietal and omental adhesions; clamp; 32 lbs.; incision 6 inches	...	"
194	"	31	"	Parietal and omental adhesions; clamp and ligature; 9 lbs. 5 oza. and 13 pints ascitic fluid; incision 10 inches	...	Died in 33 hours.
195	"	55	"	Parietal, omental, and intestinal adhesions; clamp; 15 lbs.; incision 6 in.	...	Septicæmia.



SERIES III.—Cases where an *Exploratory Incision* was made.

No.	Date	Age.	Condition.	Connections; treatment of pedicle; weight of fluid or tumour; length of incision, etc.	Result.
1	1886 Aug.	38	Married	Nineteen pints of ascitic fluid removed, and a malignant tumour exposed, involving uterus and ovaries	Relieved, but died a few weeks afterwards.
2	Dec.	39	"	Much ascitic fluid removed; solid tumour of uterus exposed and punctured	Relieved. Still alive.
3	"	43	"	Renal cyst exposed and tapped. See 'Dublin Quarterly Journal,' Feb., 1897	Death in 80 hours. Uræmia.

	Recovered.		Died.		Mortality per cent.
105 Married or Widows .....	68	.....	37	.....	35·23
95 Unmarried .....	70	.....	25	.....	26·31
<u>200</u>	<u>138</u>		<u>62</u>		<u>31·</u>

In the first 100 cases the mortality among married and unmarried women was nearly equal. A much smaller mortality has been observed among the unmarried women in the second 100 cases.

### *Social condition.*

In the first 100 cases the mortality was smaller in hospital than in private practice, the deaths averaging 29·6 per cent. in the hospital, and 39·1 in the private cases. In the second 100 cases this proportion is directly reversed. There were :

			Recovered.		Died.		Mortality per cent.	
Hospital Cases .....	35	.....	23	.....	12	.....	34·2	
Private Cases .....	65	.....	49	.....	16	.....	24·6	
	<u>100</u>		<u>72</u>		<u>28</u>			

Thus in the first series there was a greater mortality of 10 per cent. in private than in hospital practice, and in the second series the greater mortality, also of 10 per cent., was in hospital practice. Taking the 200 cases there were—

			Recovered.		Died.		Per cent.
Hospital Cases	89	.....	61	.....	28	.....	30·4
Private Cases	111	.....	77	.....	34	.....	30·6
	<u>200</u>		<u>138</u>		<u>62</u>		<u>31·</u>

These results are so nearly identical that it is probable the difference in the two series is only accidental, or such as is almost certain to occur in statistical inquiries when only small numbers are dealt with, and which can only be corrected by the test of larger numbers.

I may preface some remarks upon the

### *Situation and length of the incision*

by the following table, which shows the result of different lengths of incision in 200 cases :—

the ligature left as a seton in the peritoneal cavity, and the wound has been united by sutures, which have not brought together the whole thickness of the abdominal wall.

Another operator would remove a similar tumour after emptying it or breaking it up, through an incision of 4 to 6 inches in length below the umbilicus. The pedicle would be fixed between the edges of the wound with its secured end above the skin, and the whole thickness of the abdominal wall would be carefully brought together by sutures passing through its peritoneal coat.

It is obvious that the result in the two cases may be affected by other considerations than the length of the incision. So that a mere statistical inquiry as to the results obtained by different operators by incisions of different lengths, could be of very little value—certainly of less value than an equal number of cases by the same operator.

Historically, however, the inquiry is of interest, and may be of some importance as a guide to future progress, now that a considerable number of cases by the long and short incision may be compared in the practice of one operator.

In all my cases the *linea alba* has been selected as the seat of incision, and in a very large majority of the cases on record other operators have selected the same situation. But in some few cases the incision has been intentionally carried either to the right or left of this line. One of the *lineæ semilunares* has been occasionally, though very rarely, selected; and in some few exceptional cases oblique or transverse incisions have been made. Thus Dr. Atlee<sup>1</sup> in one successful case made an incision seventeen inches long, from the symphysis pubis to the middle of the crest of the right ilium. Bühring<sup>2</sup> made an incision at the outer border of the external oblique on the right side from the false ribs to the crest of the ilium.

In one of the earliest cases in England, Mr. King<sup>3</sup> made one vertical incision seven or eight inches long to the right of the

<sup>1</sup> 'American Journal Med. Science,' 1849 and 1855.

<sup>2</sup> 'Heilung der Eierstocks geschwülste,' 1848.

<sup>3</sup> 'Lancet,' January, 1837.

firm, unyielding, and perfect a portion of the abdominal wall as the uninjured muscle in its normal state—as I do not think that division of the muscle can make union of skin, peritoneum, or cellular tissue more certain or complete—and as I never once saw any want of union when the recti had been carefully avoided, I always endeavour to divide the linea alba accurately, without opening the sheath of either rectus.

It is not often easy to do this, for the weight of the tumour has generally either drawn the recti to one side, or the muscles have been spread out over the anterior surface of the cyst. *Anatomically*, it appears a matter of some importance not to open the sheath; but although it is well to try to hit the linea alba exactly, it does not appear of much importance *surgically* if one edge of the muscle be exposed, or if a division be made through the muscle parallel with the course of its fibres. If the incision be extended above the umbilicus, it is better to carry it round to the left side, because the round ligament of the liver passes diagonally upwards and backwards towards the right side, and might be wounded if the incision were carried either directly through the umbilicus, or to the right side. In some cases a wound of the round ligament might not be of consequence, but in others it might lead to serious hæmorrhage, as the embryonal umbilical vein is not always entirely obliterated, but remains patent, and is sometimes of considerable size.

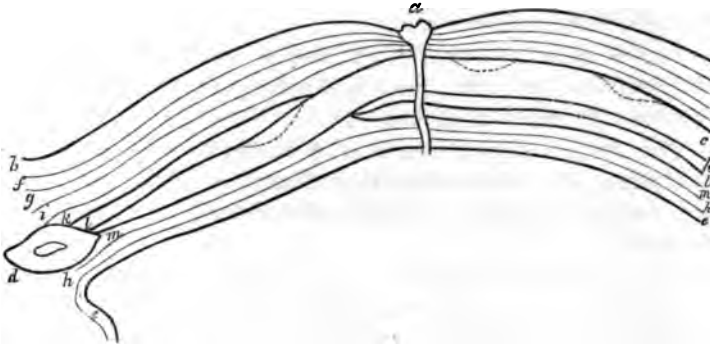
When the linea alba is chosen for the incision the following structures are successively divided :

1. The skin.
2. The subcutaneous areolar tissue, with fat of varying thickness.
3. The interlaced fibres of the aponeuroses of the abdominal muscles constituting the linea alba.
4. Layers of the fascia transversalis with more or less fat. The uppermost layer adheres closely to the linea alba. The deepest layer is only very loosely connected with the peritoneum.
5. The peritoneum.

But this normal arrangement is often much modified.

on either side of the linea alba through one of the recti muscles.

No. 2.

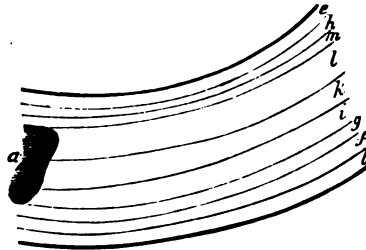


- a.* Umbilicus.
- b.* Skin.
- c.* The rectus muscle with its inscriptions tendinæ.
- d.* Symphysis pubis.
- e.* Peritoneum.
- f.* Superficial layer of areolar tissue.
- g.* Deep layer of ditto.

- h.* Perimysium internum.
- i.* Aponeurosis of external oblique muscle.
- k.* Aponeurosis of internal oblique muscle.
- l.* Aponeurosis of transversalis muscle.
- m.* Fascia transversalis.

The diagram No. 3 shows the layers divided if the incision be made along one of the lineæ semilunares.

No. 3.



- a.* Crest of the ilium.
- b.* Skin.
- c.* Peritoneum.
- f.* Superficial layer of areolar tissue.
- g.* Fascia superficialis.
- h.* Perimysium internum.

- i.* Aponeurosis of external oblique muscle.
- k.* Aponeurosis of internal oblique muscle.
- l.* Aponeurosis of the transversalis muscle.
- m.* Fascia transversalis.

under the vein. In this way, what might be otherwise a serious loss of blood is prevented.

3. *The sheaths of the recti*, complete anteriorly, incomplete posteriorly, from about two inches below the umbilicus, formed by the aponeurosis of the flat abdominal muscles, and terminating in the linea alba, hardly require more than a passing mention. But if much disturbed during the first incision, abscess is very likely to delay healing.

4. *The recti and pyramidales* muscles are almost always seen, and one or other is generally divided in ovariectomy. When the recti are unusually broad near the pubes, the pyramidales may be absent. When the recti are narrow below, the pyramidales lying in front of the recti, and inclosed in the sheath, are inserted into the inner border of the sheath, half-way between the pubes and the umbilicus, or even higher.

5. The fibres of the flat abdominal muscles cross each other in different directions, embrace the recti muscles, and conjoin on the *linea alba*, forming a tendinous band, which is very strong at the pubic end, and broader and weaker at the sternal end. The fibres of the aponeurosis on one side continue across the linea alba, and interlace with fibres coming from the opposite side, forming meshes which in the normal state are very small, only giving passage to nerves and vessels; but which, after great distension of the abdominal wall, form apertures through which small masses of fat may escape from beneath, forming what have been called *Hernie adiposæ*, and often leading an inexperienced ovariectomist to think that he has opened the peritoneal cavity, and exposed the omentum.

6. The *umbilicus* is merely one of these openings in the linea alba; but the occasional permeability of the embryonal umbilical vein (already referred to) must be borne in mind, and the fact that the urachus may also remain permeable, and urine escape from the bladder through it at the umbilicus. I have never seen this in the adult; but in one case of ovariectomy I found the urachus, though closed at both ends, open for the whole length of my incision in the abdominal

REPORT  
OF  
THE SCIENTIFIC COMMITTEE  
APPOINTED TO INVESTIGATE THE  
PHYSIOLOGICAL AND THERAPEUTICAL EFFECTS  
OF THE  
HYPODERMIC METHOD OF INJECTION.

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THE Committee in the following report having to choose between the two terms Subcutaneous and Hypodermic, have resolved to employ the former, as no advantage seems to them to be gained by the substitution of the latter term ; at the same time they do not wish it to be understood that they object to the term Hypodermic.

In drawing up their Report on this method of introducing drugs into the body, the Committee cannot but express how conscious they are that their experience in many points, and especially in the therapeutical portion of the subject, is limited ; and in so wide a field of investigation they do not hope to exhaust the many points of interest which have suggested themselves during the inquiry.

The conclusions that have been made on this subject have been drawn from three sources of information. These are :—

- 1st. Original experiments on animals and on man in health and disease.
- 2nd. Personal evidence of experienced medical men given before the committee.
- 3rd. Records of facts and other communications received

Selection was made of drugs, small in bulk and intense in effects, and a clear watery solution was the general form used.

The following alkaloids specially employed the attention of the sub-committees :

Aconitine,  
Atropine,  
Morphine,  
Strychnine,  
Quinine.

Experiments were also made with the following substances :

Calabar bean,  
Conia,  
Hydrocyanic acid,  
Iodide of Potassium,  
Podophyllin,  
Colocynth,  
Aloes,  
Battley's solution of opium.

Great care was taken to obtain pure drugs for experiment. Messrs. Morson, of Southampton Row, supplied the solution of the various alkaloids; Bell and Co. supplied the extract of Calabar bean; Hanbury, of Plough Court, the solutions of Podophyllin, Colocynth, and Aloes; and the thanks of the committee are due to Mr. Cornelius Hanbury for his courtesy and liberality in preparing and presenting these solutions.

The method of injection adopted in these experiments was, to raise a fold of the skin between the fingers and introduce the solution into the cellular tissue beneath the skin.

This practice was adopted to secure uniform results in the various experiments.

*Injecting Syringes.*—The most useful forms of syringe for subcutaneous injection are, the simple piston syringe made



appeared to be little impediment to the injection of a much greater quantity in this locality; but from experiment 2 it will be seen that a very small quantity of water injected under the skin of the thigh (at the outer side) gave rise to much tenderness and redness, these effects not being entirely removed for three days.

Hence it would appear that the quantity of fluid tolerated when injected subcutaneously varies directly as the yielding and elastic quality of the skin at the locality injected, and the most suitable places for injection are those where the skin is thin and movable.

In using drugs which require an acid to render them soluble in water, it was found that very acid solutions are apt to irritate, and the solutions were therefore carefully neutralized.

Very alkaline solutions should also be avoided for the same reason.

The various local effects which were found to result from the injection of certain drugs will be mentioned in their several places: the simplest watery solutions were used, other menstrua (as chloroform and alcohol) being precluded on account of the local irritation found to follow their use.

To some it may seem probable that in the operation of subcutaneous injection, in thus thrusting somewhat blindly the syringe beneath the skin, a vein may be often pierced and the drug be introduced directly into the circulation; but the risk of a vessel of any size receiving the fluid injected may fairly be argued to be at least remote, for notwithstanding the numerous experiments made by the committee, no unexpected or dangerous results have occurred, though, to judge from the amount of hæmorrhage occasionally observed on withdrawing the syringe, veins have been transfixed.

With ordinary care large superficial veins may be generally avoided; and this ought always to be taken, for injection into a large vein may produce dangerous effects.

Experiments were made for the purpose of comparing absorption by skin and vein. These are detailed at p. 590;

briefly stated the results were as follows:—The injection of one grain of the acetate of morphine into the femoral vein of a rabbit produced almost instantaneously very intense narcotism: injection of the same dose into the cellular tissue of another produced symptoms of absorption only after eight minutes had elapsed, and the effects were not so intense as in the former case.

It is worth mentioning that in performing the first experiment a great deal of care was necessary to prevent perforation of the walls of the vein, and to insure the reception of the total quantity of the fluid injected. In a subsequent endeavour to repeat the experiment, there was complete failure, owing to the syringe traversing the vein. The probability of accidentally injecting into the canal of a vein may be therefore looked upon as remote.

The pain of puncture is generally inconsiderable, but the needle should be passed suddenly through, and at right angles to the surface of the skin, in order that the cellular tissue may be reached as shortly as possible; the density of the skin influences the amount of pain; the less the resistance presented to the needle, the less the pain experienced on puncture.

The arm near the elbow offers a convenient locality for injection.

*Aconitine*.—From the expense attending the manufacture of this drug, perfectly pure samples are difficult to obtain. The solution with which the following experiments were made was prepared expressly by Messrs. Morson; two drachms of the solution contained one grain of the alkaloid.

The effects produced by aconitine on rabbits differ, in kind, very slightly whether the drug be given by the mouth, skin, or rectum.

When the drug is given by the mouth, the animal begins immediately to chew and work its mouth, rubbing its lips with the paws and shunning the light. After a short interval the salivary glands become much affected, the saliva dribbling profusely from the mouth. After several severe

attacks of retching, the animal becomes subject to convulsive spasms, principally affecting the hind legs; finally, a rapid and violent scour round the room ushers in a series of convulsions, ending in death.

Symptoms of a similar character follow the introduction of the drug into the rectum, or under the skin, the chief differences worth noticing here being that the local action of the drug is exhibited in different ways; when given by the mouth, the drug affects the salivary glands immediately and most intensely; when introduced into the rectum it irritates the gut, and defecation follows; when the drug is injected subcutaneously, it gives rise to local pain.

A comparative table of the results of the experiments made with aconitine in rabbits is subjoined.

*Results of experiments with Aconitine.*

Dose in grains.	First effects.	Result.	Duration.
<b>By SKIN.</b>			
1.480th	Immediate	Recovery	5 hours.
1.480th	"	"	5 "
1.320th	"	"	4 "
1.300th	"	Death	4½ "
1.240th	20 min.	"	2 "
1.240th	Immediate	"	25 min.
1.200th	11 min.	"	53 "
<b>By RECTUM.</b>			
1.240th	No effect	No effect	—
1.160th	—	Recovery	1 hour.
1.90th	12 min.	"	1½ "
1.60th	Immediate	"	—
1.50th	—	Death	2 hours.
<b>By MOUTH.</b>			
1.120th	Immediate	Recovery	2 hours.
1.60th	"	Death	1½ hours.

From some other experiments made with a different sample of aconitine supplied by Messrs. Morson, far less active in its effects than the solution subsequently supplied,

a theory was suggested that aconitine might lose its efficacy with age.

Other experiments were, therefore, made eight months after those just detailed with the same solution, and it was not found that its vigour was enfeebled.

The following were the results obtained by injection beneath the skin.

Weight of rabbit.	Dose in grain.	Result.	Duration of effects.
4 lb. 4 oz.	1-210th	Death	30 min.
3½ lb.	1-400th	"	1 hour 16 min.
4 lb.	1-450th	Recovery	3 hours.
Same rabbit as above, 24 hours after.	1-450th	Death	5 "

*Atropine.*—The solution of this drug was made by dissolving atropine with a small quantity of sulphuric acid, and diluting with water until 3j of fluid contained one grain of atropine.

This drug is readily tolerated by animals, and no decided results were obtained by a comparison of its effects on rabbits and guinea pigs by the three methods. From repeated experiments on those animals the effects appeared to be similar as to time of absorption and severity of effects; a quarter of a grain of atropine seemed to have no effect when injected subcutaneously; half a grain very slight; one grain a transient effect; two grains were sometimes but not always fatal. The effects of the drug by the three methods were dilatation of the pupils; torpor; loss of power in the hind limbs; much thirst; the animals lying on their sides in a semi-comatose condition.

In man, the drug acts very peculiarly when injected under the skin.

The ordinary symptoms of atropine (taken by the mouth) are well known. Briefly stated, they are dryness of the fauces; dilatation of the pupils; retardation of the pulse. These symptoms follow the administration of the drug by

the rectum. There is also an increase of temperature observed in both cases. When atropine is given subcutaneously, there is a preliminary stage of excitement which is a remarkable characteristic of this method.

The symptoms of the drug (given by the skin) are as follows:—After the injection some local tingling without much evidence of irritation generally occurs; the duration of this local effect being from a quarter of an hour to some hours; at a time varying from 4—10 minutes after the injection, a sudden, rapid, and unmistakable acceleration of the pulse can be perceived, with an alteration of quality readily felt by the finger, synchronous generally with a feeling of dizziness and pain across the forehead. The number of respirations is increased and the temperature of the body raised, a general sensation of warmth and flushing of the face accompanying these phenomena. The pupils subsequently begin to dilate; great dryness of the throat and mouth is experienced and the patient occasionally suffers from difficulty in emptying the bladder; the stage of excitement passing off gives way to gradual decrease of temperature, and in the rapidity of the pulse and the respirations. If the dose be large a peculiar kind of restless delirium is observed.

The following are the results of experiments on a man (æt. 58):

Dose 1-40 gr.	Absorption.	Effects.	Duration.
Mouth.... ..	25 min.	Dilatation of pupils. Pulse, rate of respiration, and heat increased. Dilated pupils. Dryness of throat.	1 hour.
Skin .....	8 min.		6½ hours.

The next table shows the effects produced in a boy (æt. 6). But it must be noticed that the experiment by the rectum followed on the day after the drug had been taken by the mouth.

Dose 1-60 gr.	Absorption.	Effects.	Duration.
Skin.....	10 min.	Pulse accelerated 24 beats. Pupils widely dilated.	4 hours 15 min.
Mouth.....	1 hour 30 min.	Pulse accelerated 8 beats. Pupils widely dilated.	Uncertain.
Rectum .....	Uncertain	Pupils slightly dilated.	4 hours 15 min.

The following experiments were made on a man (æt. 26).

Quantity 1-160 gr.	Effects.	Duration.
Skin.....	Temp. increased 1.2°. Pulse increased 26 beats. Large pupils. Throbbing of head and dryness of throat. Respirations diminished.	1 hour 20 min.
Mouth.....	Temp. increased 1.1°.	—
Rectum .....	Temp. increased 1.3°. Pulse increased 4 beats.	—

In a woman (æt. 24) the following results were obtained.

Dose 1-160 gr.	Effects.	Duration.
Skin.....	Temp. increased 1.2°. Headache and dryness of throat.	1 hour 20 min.
Mouth.....	Temp. increased .6°.	—
Rectum .....	Temp. increased 1°. Throat dry.	—

*Morphine.*—This drug was dissolved by rubbing up the acetate of morphine with just sufficient dilute acetic acid to render it clear, adding hot distilled water until 3j of the solution contained gr. x of the acetate. The solution should be carefully neutralized with liquor potassæ, as its acidity is apt to give rise to troublesome irritation. No other local effects but slight and transient tingling have been found to follow the injection of the neutral solution. Different samples of this drug, even from the same laboratory, are found to vary in the amount of acid required to make a clear solution; the colour of the solution when first

made should resemble that of pale sherry; the solution becomes darker and acid by keeping.

Rabbits tolerate such large doses of this drug that many experiments were not made. Subjoined are the results of some experiments made on two full-grown and equal-sized rabbits. The symptoms, though varying in intensity by the three methods of administration, were not found to vary in character.

One of the early symptoms of absorption is contraction of the pupils; the pulse is increased in rapidity, and narcotism follows, the animal lying with its legs sprawling.

The various doses with their effects are given below.

Dose in grains.	Absorption.	Symptoms.	Result.	Duration.
<b>By SKIN.</b>				
1	8 min.	Complete coma and paralysis	Recovery	2 hours.
1	8 min.	"	"	2½ "
<b>By RECTUM.</b>				
1	35 min.	Slight drowsiness	Recovery	45 min.
2	6 min.	"	"	34 "
2	4 min.	"	"	15 "
<b>By MOUTH.</b>				
1	—	—	—	—
2	—	—	—	—
2	—	—	—	—
5	7 min.	Slight drowsiness	Recovery	30 min.
5	7 min.	"	"	30 "

It is almost superfluous to say that much smaller doses are necessary for human beings; indeed there are instances, though such are happily rare, where dangerous results have followed the subcutaneous injection of a quarter of a grain of morphine. The general type of symptoms appears to be similar, whether this drug be given by the mouth, the rectum, or the skin; there are certain differences, especially in the relative degree of certain symptoms, but that there is no distinctive action assignable to one or the other method

(as some have supposed) is shown by similarity in the order of symptoms, and further strongly supported by the fact that peculiar idiosyncrasies exhibited by some individuals with respect to this drug, when taken by the mouth, have been known to follow its subcutaneous injection.

The ordinary effects of the drug, when taken by the mouth, are recorded here for the sake of comparison with those following the subcutaneous injection.

The train of symptoms from one sixth of a grain by the mouth may be given in the following order:—The first symptom was nausea, which was accompanied, in twenty-five minutes, by tightness across the brows and headache; the pulse was retarded; the saliva diminished; the mouth and fauces became dry; the pulse was still farther retarded the nausea passed off in a few hours, the most persistent symptom being the headache.

With larger doses the pupils become contracted, and the nausea and loss of appetite may continue for some time after the other symptoms have passed off, or vomiting may even occur.

Nausea was not observed to follow the introduction of the drug into the rectum in so marked a degree as when it was given by the mouth; in some cases, itching of the skin was a prominent symptom, but no other difference in the kind of symptom was observed.

The following symptoms were experienced from the injection of one sixth of a grain into the cellular tissue:—The first signs of absorption were dizziness and faintness (the period of absorption varies from 4—10 minutes); then ensued headache; nausea; acceleration of the pulse with slight increase of temperature; burning heat of the face and flushing of the cheeks.

The period of excitement passes off in (about) twenty minutes; the pulse is retarded; the temperature falls; the natural secretions are diminished; the saliva ceasing to flow, the mouth and fauces become dry and constricted; general lassitude and a disposition to sleep come over the patient; the pulse beats more slowly, and occasionally it



irregular; in some cases itching of the skin is felt, in others profuse sweating.

If the dose be large, retching and even vomiting may follow, nausea and headache being prolonged for some time.

The following table shows the results of some experiments in a healthy man (æt. 32). One sixth of a grain of the acetate of morphine was used in each instance.

Symptoms.	Skin.	Mouth.
First absorption .....	5 min.	110 min.
Pulse increased .....	8 beats	None.
Pulse lowered .....	12 "	10 beats.
Headache .....	36 hours	10 hours.
Nausea .....	46 "	3 "
Pulse recovered its natural standard	22 "	8 "
Incapacity to work .....	6 hours	None.
Total duration of symptoms .....	46 "	11 hours.

The objective symptoms from morphine are not so remarkable as those from atropine, and trustworthy data of the effects of morphine are consequently difficult to obtain.

The following are some of the results obtained from experiments on man.

Man, æt. 50.

Skin .....	$\frac{1}{2}$ gr.	Acceleration of pulse, followed by retardation; intense itching of skin, profuse sweating, drowsiness.
Mouth ...	$\frac{1}{2}$ gr.	Slight diminution of pulse, 4 beats per minute.
Rectum ...	$\frac{1}{2}$ gr.	Considerable diminution of pulse, 14 beats per minute.
Skin .....	$\frac{1}{2}$ gr.	Retardation of pulse 12 beats; loss of temperature $8^{\circ}$ ; respirations diminished 8. Flushes, sweats, dryness of throat.
Mouth {	$\frac{1}{2}$ gr.	No effect.
	$\frac{1}{2}$ gr.	Retardation of pulse 4 beats; loss of temperature $1^{\circ}$ . Slight drowsiness.
Rectum ...	$\frac{1}{2}$ gr.	Retardation of pulse 7 beats. Itching of the skin.

## Man, æt. 28.

Dose 1-gr.	Absorption.	Symptoms.	Duration.
Skin .....	6 min.	Pain across forehead, dryness of throat, flushes and intense headache, sleep.	9 hours.
Mouth ...	7 ..	Pain across forehead not severe, nausea, drowsiness.	3 ..

It should be mentioned that in these as in the other experiments recorded here, care was taken that the conditions of the individual under experiment should be similar; due attention being paid to the state of the bowels, and the drug being given at the same hour on each day of experiment.

*Quinine.*—This drug was dissolved by rubbing up with sulphuric acid in the smallest possible quantity, and adding water until gr. v of quinine were dissolved in 3j of fluid.

Experiments on animals were not made with this drug; toxic effects not being its prominent characteristic.

Some interesting results were obtained from the experiments made on the human subject. It was not found that any local effects followed the injection of the solution in a healthy individual so long as the quantity of water in which it is dissolved is less than half an ounce; some redness and tenderness followed the injection of three grains with ʒss of water in one case, and in another an abscess, which had to be opened, formed from the injection of five grains in an ounce of water.

When the drug is injected into the cellular tissue, considerable elevation of temperature and acceleration of the pulse with headache are the symptoms observed; when the drug is given by the mouth or rectum the symptoms are apparently of a similar nature, but are less marked; the difference between the skin and the mouth being more marked than between the skin and the rectum.

The increased heat of skin and quickening of the pulse, which are caused by five grains of quinine given by the

skin, are slight or inappreciable when the same dose is taken by the mouth.

A comparison of the various methods is presented in the following tables :

Dose 3 grs.	Absorption.	Effects.	Duration.
Skin .....	35 min.	Increase of temperature. Accelerated pulse.	2½ hours.
Mouth ...	—	—	—
Rectum ...	45 min.	Increase of temperature.	1½ hour.
Skin .....	4 min.	Temperature increased. Pulse accelerated. Headache considerable.	29 hours.
Mouth ...	60 min.	Temperature increased.	3·45 „
Rectum ...	40 min.	Temperature increased. Pulse accelerated. Headache.	6·30 „
Dose 5 grs.			
Skin .....	8 min.	Increased heat. Quick pulse. Headache.	1·10 hour.
Mouth ...	—	Increased heat. Slight frontal headache.	Uncertain.
Rectum ...	10 min.	Increased heat. Quick pulse.	1·25 hour.

As the increase of temperature is a most remarkable effect of quinine, the subjoined table has been given of the relative increase in degrees (Fahrenheit), by these three methods.

Dose in grs.	Skin.	Mouth.	Rectum.
3	1°	—	$\frac{8}{10}$ °
3	$2\frac{7}{10}$ °	$1\frac{1}{10}$ °	$1\frac{1}{10}$ °
5	$\frac{4}{10}$	—	—

*Calabar bean*.—Some experiments were made on rabbits, with an alcoholic extract of the bean (prepared by Messrs. Bell),  $\text{mij}$  of the extract being equivalent to gr. iv of the powdered bean. The extract becomes milky when water is added, but it is otherwise too viscid to pass through the syringe.

The symptoms of this drug, manifested after the subcutaneous injection, were general tremors, the animal crying as with pain and rushing wildly about, knocking against things that stood in its way; this was followed by salivation, convulsions, paralysis of the hind legs, slight contraction of the pupils; the respiration became first more rapid, and finally slower, death setting in after convulsions; the heart continued to beat after the respirations had ceased; the pupils after death dilated, and then contracted.

The lungs were found to be bloodless, and both cavities of the heart were full of dark blood.

$\text{mij} = 12$  grains of the bean, were administered to three full-grown rabbits, as shown by the following table :

	Quantity.	First symptoms.	Effects.	Duration.
Skin .....	$\text{mij}$	In 9 minutes, tremors and paralysis.	Paralysis. Recovery. No effect on pupil.	62 min.
Mouth ...	"	No effect	—	—
Rectum ...	"	"	—	—

*Conia*.—A solution of this drug was made by the addition of a small quantity of dilute sulphuric acid and of water, so that  $\text{zij}$  contained four grains of the alkaloid.

When the drug was given subcutaneously to rabbits, the following symptoms were observed. The animal moved heavily, and, in a short time, showed symptoms of paralysis affecting the hind legs; it was unable to maintain equilibrium of the hinder quarters, and it frequently backed against the wall for support; the respirations became

rapid and irregular; intense sensibility of the hind quarters was exhibited, the animal starting even if its fur was moved by the breath. Eventually it was seized with convulsions, the back being arched forwards (emprosthotonos), the respirations becoming slow, while the pulse became more rapid and irregular.

During this time the animal seemed to be sensible; at length the respirations ceased altogether, the heart beating for some time afterwards. The pupils were found to contract after death.

By the mouth the symptoms were similar in character, but retching and vomiting were prominent characteristics of this method of introducing the drug.

By the rectum a similar train of symptoms was exhibited, with the exception that retching was not observed.

The following table shows the results obtained:

Quantity in grs.	Absorption.	Effects.	Duration.
By SKIN.			
$\frac{1}{4}$	19 min.	Slight	30 min.
$\frac{1}{2}$	17 min.	Severe	3 hours.
$\frac{3}{4}$	6 min.	"	2 "
$\frac{1}{2}$	5 min.	Death	22 min.
$1\frac{1}{4}$	26 min.	"	39 "
By MOUTH.			
$1\frac{1}{4}$	—	None	—
3	$1\frac{1}{4}$ hour	Slight	—
4	8 min.	Severe	30 min.
5	36 min.	Death	$2\frac{1}{4}$ hours.
By RECTUM.			
$\frac{1}{4}$	—	None	—
1	20 min.	Slight	—
2	38 min.	"	—
3	10 min.	"	2 hour 40 min.
4	50 min.	"	—
5	17 min.	Death	1 " 34 "

*Strychnine.*—A solution was made by rubbing up the alkaloid with a little phosphoric acid, and adding water until 3ij contained one grain of strychnine.

The experiments were directed to the investigation of the poisonous effects on rabbits by the three methods; the subjoined tables show the minimum dose that would kill when the drug was given by the mouth, the rectum, and the skin.

Dose in gr.	Absorption.	Effects.	Duration.
By SKIN.			
1-30th	2 min. 30 sec.	Death	7 min.
1-60th	3 min. 10 sec.	"	5 "
1-120th	6 min. 45 sec.	"	22 hours.
1-120th	4 min. 30 sec.	Recovery	—
1-180th	—	—	—
1-180th	—	—	—
By MOUTH.			
1-30th	5 min.	Recovery	10 min.
1-20th	—	—	—
By RECTUM.			
1-20th	15 min.	Death	20 min.
1-25th	12 min.	"	32 "
1-30th	—	Recovery	—

A second series of experiments was made to determine the comparative rapidity of absorption.

Dose in gr.	Absorption.	Result.	Duration.
By SKIN.			
1-8th	2 min. 30 sec.	Death	5 min.
1-16th	3 min.	"	4 min. 45 sec.
By MOUTH.			
1-16th	21 min. 40 sec.	Death	23 min.
By RECTUM.			
1-8th	5 min. 30 sec.	Death	7 min. 30 sec.
1-16th	5 min. 15 sec.	"	7 min.

From these tables it will be seen that the smallest fatal dose was  $\frac{1}{120}$  gr. by the skin;  $\frac{1}{16}$  gr. by the mouth;  $\frac{1}{8}$  gr. by the rectum.

*Hydrocyanic acid.*—A series of experiments was made in a similar manner with hydrocyanic acid.

The following is a table of the results :

Dose.	Absorption.	Effects.	Duration.
By SKIN.			
$\text{m} \frac{1}{4}$	2 min. 30 sec.	Death	3 min. 30 sec.
$\text{m} \frac{1}{2}$	1 min. 30 sec.	"	5 min.
$\text{m} 1$	—	—	—
By MOUTH.			
$\text{m} \frac{1}{4}$	—	—	—
$\text{m} \frac{1}{2}$	—	—	—
$\text{m} 1$	1 min. 20 sec.	Death	1 min. 45 sec.
$\text{m} 1$	1 min.	"	1 " 30 "

*Iodide of potassium.*—The following series of experiments was made on a healthy man, who had a congenital extroversion of the bladder. He was dieted and kept in bed during the experiments, but the series (as will be seen on looking at the details) was not completed, owing to the disagreeable local effects of the drug.

The object of the experiments was to ascertain the relative rapidity of absorption and excretion ; computation being made from the time at which the drug was introduced into the system to the time of its detection, by the starch test, in the urine.

No peculiar symptoms were observed during the passage of the drug, after any of the methods of administration ; but the subcutaneous injection caused much local irritation, and eventually a small ulcer was formed at the seat of the puncture.

The following were the results obtained :

Dose, in grs.	Period after a meal.	Time of passage.
By SKIN.		
1	1½ hour	20½ min.
By MOUTH.		
4	7 hours	14½ min.
4	4 "	14½ "
4	1½ hour	41½ "
By RECTUM.		
4	4½ hours	23 "
4	4 "	—
4	4 "	—

*Podophyllin*.—A solution of this drug was made with equal parts of liquor potassæ and water. When the drug is pure, the resulting solution is a very convenient form for injection, and it will bear the addition of any quantity of water without precipitation. Some samples of this drug were found to consolidate into a gelatinous mass under the above treatment; this property belongs to the paler sort.

A solution containing gr. j of podophyllin in ℥x, was found to cause a considerable amount of local irritation, but on diluting the solution so that gr. j was contained in ℥xviii, the fluid was found to be innocuous. A comparison of the results obtained from experiments on healthy men is given in the following tables.

A peculiar characteristic of the subcutaneous injection of this drug was, that free diuresis was caused in about twenty-four hours after purgation.

Dose, ʒs gr.	Effects.	Time.
Skin .....	Copious motion in .....	2 hours.
Mouth .....	Purgation in .....	9 hours and 18 hours.
Rectum... {	Action of bowels in ...	3 hours.
	Purgation in .....	18 "



Dose in grs.	Locality.	Effects.	Time.
$\frac{1}{8}$	Skin .....	Free purgation in .....	6½ hours.
$\frac{3}{10}$	Mouth .....	and again in .....	13 "
$\frac{3}{10}$	Rectum ...	Copious purgation in...	18 "
		Slightly in .....	8 "
		Freely in.....	15 "

Dose in grs.	Locality.	Effects.	Time.
$\frac{1}{8}$	Skin.....	Purgation in .....	9 hours.
$\frac{3}{10}$	Mouth .....	and again in.....	13 "
$\frac{3}{10}$	Rectum ...	Purgation in .....	11 "
		and again in.....	18 "
		Purgation in .....	9 "
		and again in.....	12 "

*Aloes*.—The watery extract of aloes was diluted with an equal quantity of distilled water, and the solution thus obtained was used for injection. Every attempt to render aloin soluble failed. The above solution was thought to be the best for use, as this mode leaves the drug in its natural condition, but the solution is distinctly acid.

The subcutaneous injection of this fluid gives rise to considerable local tenderness and swelling, with general *malaise* and restlessness. No stool was produced until twenty-four hours had elapsed, when a scanty one was passed. The arm injected was swollen, tender, and painful on motion, for three days after the operation. The symptoms produced were so unfavorable that no further experiments were made.

Mr. C. Hanbury has ascertained that when the solution is neutralized by liquor potassæ, there is no disposition to deposit solid matter on the addition of water; perhaps such a solution might not occasion so much local irritation.

*Colocynth.*—Mr. C. Hanbury provided an almost neutral solution of the watery extract of colocynth, by dissolving in water with the addition of a little spirit, so that ʒj contained twelve grains of the extract.

From the local effects produced by injection, the drug was found to be unsuitable for this method.

A reference to the detailed experiments made with the drug will show that they involved much risk, and that considerable amount of pain was endured, such indeed few would care to encounter.

Subjoined is a table of the results.

Dose in grs	Locality.	Effects.	Time.
$\frac{1}{2}$	Skin .....	Peristaltic action in .....	3 hours.
		Motile stool in .....	12 "
		Great local irritation for ..	3 days.
$\frac{1}{2}$	Mouth .....	Vermicular action and stool	Immediate.
$\frac{1}{2}$	Rectum ...	Quiet stool in .....	2 hours.
		Abundant diuresis in .....	6 "

## THERAPEUTICAL PORTION.

In this portion also of their report the Committee have drawn their conclusions as to the therapeutical advantages that distinguish the subcutaneous method of injection from experiments with a few active medicines, and though the list might have been extended, it must be borne in mind that many valuable drugs cannot be used in this way, on account of the irritating properties they possess.

The intensity and the rapid sequence of effects which have already been shown to characterize the hypodermic method of administering drugs are important advantages, which are readily appreciated by the patient; and the dread which the slight operation may have caused at first is soon overcome when once the resulting benefits have been experienced.

In the relief of pain this method of introducing anodynes offers superior advantages to those in ordinary use; and in cases of delirium, of mania, and of tetanus, where there is resistance or impediment to the ordinary methods of administering remedies, subcutaneous injection secures not only quickness of action, but also certainty as to the introduction of the drug.

Much difference of opinion exists on the question of localising the injection. Cases have been communicated to the committee from which the superiority of local injection has been maintained; but although they have performed many experiments in reference to this question, the committee have failed to obtain any evidence to show that the local predominate over the general effects; they must therefore express their opinion, that though no symptom results from injection at the part affected, which is not shared equally by injections at any other part of the body, yet practically it may be advantageous to localise the injection

for the sake of those effects upon the mind which localisation will sometimes produce.

Injections may be repeatedly practised in the same locality without any serious or permanent injury to the part. Mr. Roberts injected himself many times successively, in a very limited area, without any worse result than temporary thickening and irritation.

The committee have endeavoured to procure details of untoward results following subcutaneous injection, and the details of these cases will be found in the Appendix. One or two other cases have come to their knowledge, but of these they have been unable to obtain any satisfactory account.

The following are the results of their experiments on man in disease with aconitine, atropine, morphine, quinine, and strychnine.

*Aconitine.*—This drug was tried in three cases of neuralgia, but the local tingling which followed the injection was so severe that the drug was considered unfit for subcutaneous use. In one case in which the neuralgia was of an hysterical character the pain was relieved; in the other two cases no alleviation was experienced. In the first case  $\frac{1}{100}$ th grain was used; in the others,  $\frac{1}{320}$ th grain, and  $\frac{1}{256}$ th grain.

*Atropine.*—The anodyne properties of this drug are exhibited in a marked degree by subcutaneous injection.

In cases of simple neuralgia, atropine when thus administered is a very valuable remedy, and, in some cases, where morphine procured only temporary relief, the benefits derived from atropine injections were permanent. Very decided results were observed to follow minute doses of the drug used in this manner.

The pulse was accelerated to a considerable degree in one case, when  $\frac{1}{180}$ th grain only had been injected. A larger dose should be given in cases of severe neuralgia, and the most satisfactory results were found to follow when decided toxic effects were manifested.

The discomfort (the excitement, the dry mouth, and the occasional disagreeable action on the bladder) experienced during the action of this drug presents a considerable hindrance to its general use. The cases in which atropine was used with advantage were cases of local neuralgia, lumbago, and sciatica.

The initial doses are the eightieth of a grain for a woman, and the sixtieth for a man, but in cases of severe neuralgia, larger doses may be given with safety. The largest dose mentioned to the committee was one tenth of a grain.

*Morphine.*—The value of this drug is materially enhanced by this method of administration, and its action is not only secured with greater intensity and rapidity than by the ordinary modes, but the duration of its effects is prolonged, and some patients can tolerate it far better when it is injected under the skin than when it is given by the mouth.

Injected subcutaneously this remedy does not invariably lose its virtues by repetition, and instances have come to the notice of the committee where the injection has been repeated daily for a number of years, without the dose being augmented. Mr. Roberts expressly states that, though the injections were repeated in his own person more than a hundred times, the dose was never increased beyond two thirds of a grain, and a smaller quantity was often found sufficient.

To confirmed opium-eaters this method has been found of much service, smaller doses than those previously taken by the mouth being requisite. The largest dose mentioned to the committee was given to such a patient; as many as eight grains of the acetate were injected in this case.

Patients suffering from cancer have derived much benefit from the use of subcutaneous injections. Mr. Reeves mentions that from six to eight grains were injected in one case daily for a considerable period.

In allaying pain, the virtues of the drug are decidedly

increased by injection, though the effects are not always permanent.

In cases of delirium tremens this method is often extremely useful, and in some instances was found to succeed where the introduction of the drug by the mouth failed; in a few instances, however, it seemed to have a negative result.

From the few cases of mania treated by injection that have come under notice, it would seem that this method of giving morphine is not altogether free from danger; in one case of mania the injection of half a grain proved fatal, and the same dose narcotized another patient for four days.

The initial dose for an adult man, under ordinary circumstances, is from one sixth to one fourth of a grain; for a woman it should be smaller, from one eighth to one sixth.

A few other cases where alarming symptoms have arisen from the injection of morphine have been forwarded to the committee, the details of which will be found in the Appendix.

Briefly stated they are the following:—

One quarter of a grain in a man, not fatal.

Twenty-five minims of the liquor morphiæ acetatis, equivalent to five twelfths of a grain of morphine, produced narcotism in a man, not fatal.

A quarter of a grain in a young lady twenty-four years of age, not fatal.

In those cases of mania already alluded to:—

Half a grain in a woman suffering from acute mania, not fatal.

Half a grain in a similar case, fatal.

In some hospitals it has been the practice to inject a small dose of morphine after operations, for which chloroform has been used; the injection being made before the effects of the chloroform have passed off. It was stated that the sleep is prolonged by these means, and the after effects of chloroform prevented, but from the experience of the

committee on this point it would seem that the sickness following the use of chloroform is not always prevented by morphine injections, though it may be retarded.

*Quinine.*—The advantages possessed by this mode of giving quinine, in the treatment of intermittents, over the ordinary method, are well illustrated by the case detailed in the Appendix. By reference to this it will be seen that the remedy can be given so as to cut short all the symptoms of the fit, even when the increasing temperature has shown its accession, and this is done in the most complete manner, which is not always the case when the medicine is given by the mouth. No local injury followed the injection of five grains of quinine in this instance; but in another, where a larger quantity of water was used, an abscess formed, perhaps from the fluid having been too rapidly injected.

Not only, then, are rapidity of action and completeness of result advantages belonging to this method of exhibiting quinine, but also economy of material—a considerable recommendation to medical men on foreign stations, and to travellers in countries where intermittents prevail and the remedy is scarce.

*Strychnine.*—This drug was injected in a few cases of paralysis, but no peculiar advantage was observed to follow its administration by the skin.

Dr. Beigel, in his evidence before the committee, mentions one case which yielded to this method; and Mr. Charles Hunter also expressed his opinion favorably with regard to the subcutaneous use of the drug.

In those cases of paralysis in which strychnine was used, the initial dose (for both sexes) was  $\frac{1}{80}$ th of a grain, and this was increased gradually to the  $\frac{1}{40}$ th of a grain.

*Podophyllin.*—No advantages seem to be gained by administering this purgative by the skin beyond those of rapidity of action and smallness of dose, nor is it probable

that this method of giving purgatives will supersede those in ordinary use.

The subcutaneous injection of the drug is sometimes followed by irritation, and in one case an abscess was formed.

The cases in which this medicine was used have been recorded in detail, but present no special features for comment.

#### CONCLUSIONS.

The conclusions which the Committee deduce from their investigations are—

1. That as a general rule only clear neutral solutions of drugs should be injected, for such solutions rarely produce local irritation.

2. That whether drugs be injected under the skin, or administered by the mouth or rectum, their chief physiological and therapeutical effects are the same in kind, though varying in degree, but—

3. That symptoms are observed to follow the subcutaneous injection of some drugs, which are absent when they are administered by the other methods; and, on the other hand, certain unpleasant symptoms, which are apt to follow the introduction of the drugs by the mouth and rectum, are not usually experienced when such drugs are injected under the skin.

4. That as a general rule, to which, however, there may be exceptions, clear neutral solutions of drugs, introduced subcutaneously, are more rapidly absorbed and more intense in their effects than when introduced by the rectum or the mouth.

5. That no difference has been observed in the effects of a drug subcutaneously injected, whether it be introduced near to, or at a distance from the part affected.

6. That the advantages to be derived from this method of introducing drugs are—

- a. Rapidity of action.



- b.* Intensity of effect.
- c.* Economy of material.
- d.* Certainty of action.
- e.* Facility of introduction in certain cases.
- f.* With some drugs the avoidance of unpleasant symptoms.

This plan, therefore, is most likely to be preferred where very rapid and decided effects are required from drugs which are operative in small doses.

W. S. SAVORY (*Chairman*).

J. A. BOSTOCK.

W. H. DICKINSON.

ARTHUR E. DURHAM.

T. HOLMES.

J. W. HULKE.

J. A. KINGDON.

HENRY LEE.

THOMAS SMITH.

REGINALD E. THOMPSON (*Reporter*).

*Aconitine.*

Morson's solution containing gr. j to ʒij ;  $m_j = \frac{1}{100}$  gr.

Exp. 4.—Grey rabbit, young, but nearly grown, healthy and strong ;  $m_{ss}$  of solution, mixed with  $m_{ss}$  of water, making  $m_{ij} = \frac{1}{240}$  grain of aconitine, was subcutaneously introduced in the region of the loins.

The immediate effect was considerable local irritation, lasting for three minutes ; the head was twisted back alternately on each side to bite at the spot punctured ; shunned light.

Four minutes.—Yawning and retching began, and hind legs seemed feeble and unsteady.

Ten minutes.—A sudden and purposeless scour in a semi-circle, as if from rhythmical spasm of the hind legs, ending in general convulsions, which quieted down into stillness of the body, with slow respiration.

Twenty-five minutes.—Died after another slighter spasm.

A litter of four rabbits was obtained, nearly grown ; two were larger and lustier than the others.

The two larger ones were taken together (A and B).

Exp. 5.—Rabbit A.—A similar dilution of Morson's solution of aconitine, and the same dose, viz.,  $\frac{1}{240}$  gr., was injected within the anus.

No effect whatever was observed.

Exp. 6.—Rabbit B.— $\frac{1}{300}$  gr. was injected beneath the skin in the region of the loins.

Immediate effect.—Local irritation ; shunned the light ; grew torpid.

Twenty minutes.—Working of jaws ; dribbling of saliva ; retching ; hind legs feeble.

Four hours.—Seemed rapidly reviving ; sat up and breathed more freely ; refused food however, and was found dead in four and a half hours from the time of puncture.

Forty minutes.—Some spasm of hind legs.

After an hour the symptoms grew milder; it became drowsy and tame; dribbling of saliva still continued.

Ate food after two hours, and recovered.

Exp. 10.—Rabbit A.—Next morning (6th day inclusive) the rabbit was quite well.

miss of diluted solution ( $\frac{1}{320}$  gr.) were subcutaneously injected near the root of the tail.

Became drowsy, and seemed tender at the region of puncture, but presented no marked symptoms. There was at first some working of the jaws, but was quite well in four hours.

Exp. 11.—Rabbit A.—On the following morning (7th day) miiij of a diluted solution =  $\frac{1}{320}$  gr. of aconitine were injected under the skin of the loins.

Immediate effect.—Much local irritation, lasting for three or four minutes.

Eleven minutes.—Retching considerably; mouth rubbed with paws.

Fourteen minutes.—Dribbling of saliva; no impairment of the voluntary powers.

Eighteen minutes.—Strong general convulsions; fell over on side, and lay gasping for two minutes; dribbling and retching ceased; gasping continued.

Twenty-two minutes.—Sat up (still gasping) and walked lamely about.

Twenty-four minutes.—Fell over on the side again after another general convulsion; from this time there were less severe convulsions, from which the rabbit partially recovered.

Forty minutes.—Fell over after a rapid scour about the floor.

Fifty-three minutes.—Died after two or three more spasms of the hind legs.

Rabbits C and D, the smaller pair of the same litter.

EXP. 12.—Rabbit C was killed in two hours by  $\frac{1}{120}$  gr. subcutaneously injected between the shoulders.

In twenty minutes.—Retching and salivation began; the other symptoms were similar to those in the foregoing experiment. Death in two hours, after a struggle and scream.

EXP. 13.—Rabbit D.

mj of a diluted solution (containing mij of Morson's solution to mxij of water) =  $\frac{1}{420}$  gr. was injected under the skin of the belly; the animal grew timid, and shunned the light; refused food; walked lamely; gaped and retched a little at intervals: but had no salivation, and after five hours took food and recovered.

EXP. 14.—Rabbit D. Two days intervened.

The same dose injected subcutaneously between the shoulders was followed by the same symptoms; recovered.

EXP. 15.—Rabbit D. Same rabbit as before.

Next day mij of Morson's solution with mij of water =  $\frac{1}{80}$  gr. were injected within the anus.

Shunned light at once.

Twenty-three minutes.—Began to work the jaws, and the muscles of the lips and cheeks, and became torpid.

No other marked symptom; recovered.

EXP. 15 *bis*.—Rabbit D.—On the following morning the same dose in the same form =  $\frac{1}{80}$  gr. of aconitine was poured into the mouth.

Almost at once the jaws began to work.

Five minutes.—Dribbling of saliva commenced.

Thirty minutes.—Dribbling became excessive.

Thirty-five minutes.—Retching.

Thirty-seven minutes.—Convulsive spasms of hind legs.

Forty minutes.—Rapid scour about the floor; fell over on its side with the hind legs seemingly paralysed; sali-

vation ceased; from this time there were alternations of convulsions and recovery. Died in a spasm in one hour and thirty minutes.

Exp. 16.—Buck rabbit, weight 4 lbs. A solution containing  $\frac{1}{30}$  gr. of aconitine was injected into the rectum.

The animal showed the presence of irritation by licking the anus. In six minutes the hind legs were dragged, and after several attacks of retching and convulsions, it died at the end of two hours.

To confirm or rectify the experiments made in April, 1866, the following were repeated in November, 1866, with the same solutions of aconitine.

Exp. 17.—November 26th, 1866.—m of the solution of aconitine (obtained from Morson for the purposes of the committee in April last) containing gr. j in two drachms of fluid were added to mxx of water.

Of this dilution miiiss, taken up in a graduated hypodermic syringe (= m $\frac{1}{4}$  of undiluted solution *i. e.*  $\frac{1}{375}$  gr.) were injected under the skin of the back of a nearly grown buck rabbit, weighing 4 lbs. 4 ozs.

There were no noteworthy effects (save a momentary indication of soreness at the spot punctured) till eight minutes had elapsed, when there were slight hiccough and sneezing.

At the expiration of ten minutes it began to work its lips and jaws, and to paw its face.

The rabbit had remained still in the place it assumed on being released, until

Eleven minutes after the puncture, when it turned round and round three or four times, tried to be sick, and shook its head.

Twelve minutes.—Ran about restlessly; hid its face behind a curtain, and began to whine.

Thirteen minutes.—Retching increased; whining and moaning more frequently; twitching of abdominal muscles.

Fifteen minutes.—Increased twitching of abdom muscles; slow respiration.

Sixteen minutes.—Purposeless scour over the floor; h legs acting spasmodically, fore legs failing.

Seventeen minutes.—This action changed into a l spasm, which drew the stomach, sternum, and pubes togeth and arched the back. The rabbit then fell over on its si with all its limbs relaxed, gasping feebly; it lay fair moaning and feebly breathing for ten minutes, when, af another spasm of the abdominal muscles and diaphrag drawing its limbs together and compressing the visc laterally out between the ribs and haunches, it died thi minutes after the injection.

Rigor mortis set in fairly and fully about two hours af death.

Exp. 18.—November 28th.—A young doe rabbit, weig ing  $3\frac{1}{2}$  lbs.

miss, =  $\frac{1}{400}$  gr., of the same diluted solution (miss of whi killed a young buck rabbit in thirty minutes on the 26t were injected under the skin of the back.

In a little more than a minute it tried to lick the poi punctured, but after five minutes it grew torpid.

Ten minutes.—Began to work its lips, still remaini torpid, but easily startled.

Thirteen minutes.—Slight tremor of head; chewi motion of jaws and lips continued.

Sixteen minutes.—Slight retching; salivation ( dribbling, but noisy smacking of lips, from moisture mouth); working of jaws and lips increasing.

Twenty-seven minutes.—Hind legs splayed out, as if l and paralysed, then gradually dragged under body agai less working of jaws.

Twenty-nine minutes.—In attempting to move, the hi legs are left behind, while all the motion is performed l the fore legs; lies stretched out on its belly.

Thirty minutes.—Gathered up its limbs again; sat u and looked in all respects more lively and better.

Thirty-four minutes.—Crawled about clumsily; hind legs useless; chewing stopped.

Thirty-six minutes.—Purposeless and stumbling run, fore legs giving way, head falling against the wall, &c.; then fell over on the side, with limbs relaxed; slow respiration; lateral spasms.

Thirty-eight minutes.—Raised its head, and gradually twisted its fore legs under, the rest of the body remaining as before; the animal continued motionless, breathing slowly and irregularly for sixteen minutes.

Fifty-five minutes.—Drew its hind legs under again, and gradually began to move in a hobbling manner.

One hour and three minutes.—Slight spasm of hind legs, which remained stretched out.

One hour and ten minutes.—Moved about again torpidly and clumsily.

One hour and sixteen minutes.—Another spasmodic scour; fell over and died.

Muscular tremor in the muscles of the thigh continued for ten minutes or longer after the cessation of respiration.

Rigor mortis after five hours.

Exp. 19.—November 29th.—Rather less than  $m_j$  of the undiluted solution of aconitine =  $\frac{1}{450}$  gr. (about) was injected to-day under the skin of the back of a young black buck rabbit, at 3.11 p.m. by means of the same graduated hypodermic syringe used on all former occasions.

After five minutes there was one attempt to bite and lick the point punctured, otherwise there was no effect, till—

Fifteen minutes.—When the rabbit became quiet and drowsy.

Seventeen minutes.—Slight twitch of body, hardly perceptible, like a start in sleep; eyes only partially closed.

Twenty minutes.—Became lively again, but soon relapsed into quietude.

Twenty-five minutes.—Sneezed.

Twenty-nine minutes.—Head jerked forward, as if retching; lips beginning to work.

Thirty minutes.—Stagger and hiccough; working of lips (not excessive) continued.

Thirty-two minutes.—Working of jaws and lips increasing; noisy smacking of lips and tongue, from motion of mouth.

Thirty-four minutes.—Stagger; retching; startings at intervals of a few seconds; noisy smacking of mouth, and frequent swallowing; no dribbling.

Fifty minutes.—Working of jaws and lips ceased; startings less frequent; sits half-lying, dull and torpid, with eyes partly closed, heedless of external impressions; does not start or move when an object is suddenly waved before its eyes; nor does it move or show any sign of hearing loud and sudden smacks of the hands; a lucifer flashed into light and waved before it produced no effect, and yet it starts occasionally when everything is still about it.

It remained in this condition, the startings being less frequent and slighter, till one hour and a half had elapsed, when it seemed to wake up, and began to move about lazily and clumsily; refused food.

These symptoms all gradually passed away; it began to eat in three hours, and then seemed to be quite well.

Exp. 20.—November 30th.—Same young buck black rabbit, which survived the injection of a little less than  $mj$  of the diluted solution of aconitine =  $\frac{1}{450}$  gr. (about) yesterday. Weight 4 lbs.

The rabbit seems to have completely recovered; has eaten heartily all day; is frisky and well.

At 4.20 p.m. as nearly as possible the same dose, viz., a little short of  $mj$  of the same solution, = about  $\frac{1}{450}$  gr., was again injected under the skin of the back.

There was no toxic effect whatever, till twenty-five minutes had elapsed, and then only a slight shudder and sneeze.

Thirty minutes.—Began to get torpid and drowsy; had occasional slight jerks and twitches of the head and body; these continued, and at



Forty minutes, there was a slight spasm of the hind legs, which lay lax and extended when this had passed off, but were gathered up again after a minute. From this time it gradually fell into the same comatose condition as yesterday, starting occasionally; was callous to all external impressions; remained unmoved upon the clapping of hands near to it, or the flashing of a lucifer in its eyes. The respiration was slow and irregular.

At the expiration of an hour there was another slight spasm and relaxation of the hind legs, which, however, were soon drawn up again.

The torpor, with slight starts and rare spasms, as above, continued till 8 p.m., then the rabbit began to move about, and seemed likely to recover; it refused food, however, and after half an hour relapsed into the same torpor, out of which it never awoke; was found dead at 9.30 p.m., *i. e.* about five hours after puncture.

Post-mortem rigidity was complete at 7 a.m. next morning—when it set in is not known.

### *Atropine.*

Exp. 21.—May 31st.—A man, æt. 58.

10.15 a.m.—P. 59. R. 15. Temp. 97.  $\frac{1}{40}$  gr. of atropine was taken on the tongue.

10.20.—No change.

10.40.—The pupils began to dilate. No other alteration.

11.5—Pupils recovering. P., R., Temp. unaltered.

11.15.—Pupils natural. No other symptoms.

Exp. 22.—June 1st, 11.27 a.m.—P. 52. R. 15. Temp. 97.5. Injected  $\frac{1}{40}$  gr. into the left fore arm.

11.35.—P. 54.

11.42.—P. 76. R. 16.

11.48.—P. 80. R. 17. Temp. 97.8.

12.20 p.m.—P. 64. R. 16. Temp. lower.

12.30.—Extreme dilatation of the pupils; great dryness of the throat.

6.—Pupils still dilated.

• **Exp. 23.**—A boy, æt. 6, suffering from incontinence urine, was treated in the following manner:

Pulse 120. Pupils  $\frac{1}{2}$  inch in diameter.

January 4th. 12 noon.— $\frac{1}{60}$  gr. of sulphate of atropine was placed on the back of the tongue.

1.30 p.m.—P. 128. Pupils  $\frac{3}{12}$  inch in diameter.

4.15.—P. 120. Pupils  $\frac{5}{12}$  inch.

No other symptoms.

**Exp. 23 bis.**—January 5th. 11.45 a.m.—P. 120. Pupils  $\frac{1}{8}$  inch.

The same dose was given by the rectum.

2.30 p.m.—P. 112. Pupils  $\frac{1}{8}$  inch.

4.—P. 112. Pupils  $\frac{1}{8}$  inch.

No other symptoms.

**Exp. 24.**—January 17th, 11.35 a.m.—P. 104. Pupils  $\frac{1}{8}$  inch in diameter.

11.45.—Same dose injected subcutaneously into the arm.

11.50.—P. 104. Pupils  $\frac{1}{8}$  inch.

12.5 p.m.—P. 108. Pupils  $\frac{3}{12}$  inch.

12.15.—P. 128. Pupils  $\frac{4}{12}$  inch.

1.20.—P. 128. Pupils  $\frac{5}{12}$  inch.

3.45.—P. 100. Pupils  $\frac{3}{12}$  inch.

*Comparative effects of the same dose of Atropine when given by the mouth, rectum, and skin.*

**Exp. 25.**—Of a solution containing gr.  $\frac{1}{4}$  of atropine in 3ij of water,  $\text{miss} = \text{gr. } \frac{1}{100}$ , were injected into the subcutaneous tissue of the calf of the leg.

The patient was a man, æt. 26, suffering from chronic disease of the left hip-joint.

Before injection.—Temp. of the mouth, 98°0'. P. 84. R. 24. Pupils  $\frac{1}{8}$  inch in diameter.

The injection was made December 5th, at 2.35 p.m.

2.40 p.m.—Temp.  $98.8^{\circ}$ . P. 98. R. 16. Pupils same.

No sensation of the presence of the drug.

3.—Temp.  $99.0^{\circ}$ . P. 100. R. 22. Pupils slightly larger.

3.10.—Temp.  $99.0^{\circ}$ . P. 100. R. 22. Pupils larger.

No sensations caused by the atropine.

3.30.—Temp.  $99.2^{\circ}$ . P. 112. R. 23. Pupils larger; throbbing in head; dryness of throat.

4.—Temp.  $99.0^{\circ}$ . P. 96. R. 20. Pupils smaller; head and throat much better.

Exp. 26.—December 8th.—miss of the same solution, = gr.  $\frac{1}{100}$ , in  $\text{z}ij$  of water, were given by the mouth.

Before taking the dose at 2.36 p.m.—Temp.  $97.2^{\circ}$ .

P. 84. R. 17. Pupils  $\frac{1}{8}$  inch in diameter.

2.45 p.m.—Temp.  $97.7^{\circ}$ . P. 90. R. 18. Pupils same as before. No toxical effects caused by the drug.

3.—Temp.  $98.2^{\circ}$ . P. 95. R. 18. do.

3.15.—Temp.  $98.3^{\circ}$ . P. 84. R. 18. do.

4.—Temp.  $98^{\circ}$ . P. 80. R. 18. do.

Exp. 27.—December 11th.—miss of the same solution, =  $\frac{1}{100}$  gr., were given by the rectum in  $\text{z}ij$  of water.

Before injection at 2.45 p.m.—Temp.  $97^{\circ}$ . P. 76. R. 18. Pupils  $\frac{1}{8}$  inch in diameter.

3.5.—Temp.  $98.2^{\circ}$ . P. 80. R. 20. Pupils remained the same. No sensations were produced by the atropine.

3.35.—Temp.  $98.3^{\circ}$ . P. 80. R. 18. do.

4.5.—Temp.  $98.3^{\circ}$ . P. 80. R. 17. do.

Exp. 28.—A woman, æt. 24, single, and hysterical. Another solution of atropine was made, containing gr. ss in  $\text{z}ij$  of water. miss of this, = gr.  $\frac{1}{100}$  of atropine, were given in water by the rectum.

Before giving the solution at 2.50 p.m.—Temp.  $96.2^{\circ}$ . P. 94. Pupils  $\frac{1}{8}$  inch in diameter.

After giving the drug—

3.3.—Temp.  $96.8^{\circ}$ . P. 85. Pupils not altered in size. No subjective symptoms.

3.17—Temp. 97.5° P. 82 do. Throat dry.  
 3.30.—Temp. 97.8° P. 82. do. do.

Exp. 29.—December 19th.—Miss of the same solution were given in water by the mouth at 2.30. p.m.

Before taking the drug—Temp. 96°. P. 84. Pupils normal

After the administration—

3.45—Temp. 96°. P. 80 Pupils not affected. No sensations caused by the drug.

4—Temp. 96.5° P. 78 do. do.

Jan. 10.—January 1st.—A very cold day. Miss of solution 1 grain were injected into the calf of the leg. A little moisture escaped

Before injecting—

2.45 p.m.—Temp. 95.2° P. 100.

3.15—Temp. 96.4° P. 98.

4—Temp. 96° P. 98 Headache, and dryness of throat.

3.10—Temp. 96.5° P. 99. Headache increased; throat very dry

3.20—Temp. 96.2° P. 100 Headache worse; throat much parched

3.30—Temp. 96° P. 99. Headache same; dryness of throat continues.

3.50—Temp. 96.4° P. 90. The same symptoms.

4—Temp. 96° P. 90. Headache severe; mouth and throat are still very dry

### *Morphine.*

Two rabbits of nearly equal size (A and B) were experimented on in the following way:

Exp. 31.—Rabbit A.—One grain of morphine in 3j of water was given by the mouth. No effect.

Exp. 32.—On the following day, one grain of morphine in 3j of water was injected under the skin at 2.55 p.m.

The pupils contracted in eight minutes. Complete coma and paralysis were produced, the rabbit lying with its legs sprawling, without any movement or resistance on being held up by the ears. P. 106.

The first movement was made at 3.55. P. 116.

Both pupils recovered at 4.15. P. 148.

It moved about languidly at 5.

Exp. 33.—Two days after, two grains of morphine in 3j of water were given by the mouth. No effect.

Exp. 34.—One day after, two grains of morphine in 3j of water were injected into the rectum at 3.55 p.m.

In four minutes the pupils began to contract, and they were small in seven minutes. The animal became drowsy and would not eat; no paralysis, and much resistance on being held up. The pupils recovered at 4.10.

Exp. 35.—A few days having elapsed, five grains were given by the mouth at 3.12 p.m. The rabbit became drowsy in seven minutes; and the pupils were small in ten minutes. It ate at 3.24, though slightly drowsy; half an hour after the injection it both moved and ate.

Exp. 36.—Rabbit B.—One grain of morphine in 3j of water was injected into the rectum.

The animal became slightly drowsy in thirty-five minutes, but this effect passed off in ten minutes, and no further symptoms were observed.

Exp. 37.—One day after, at 3.2 p.m, one grain in 3j of water was injected beneath the skin.

In eight minutes there was contraction of the pupils, followed by complete coma and paralysis. The animal moved spontaneously at 4.9, and the pupils recovered a few minutes after.

Exp. 38.—Two days afterwards, two grains in solution were given by the mouth. No effect.

Exp. 39.—On the following day, two grains in 3j of water were given by the anus at 3.56 p.m.

Pupils slightly affected at 4.2; no paralysis; the animal was rather drowsy, but ate; these symptoms continued up to 4.30, when it recovered.

Exp. 40.—Two days after, five grains were administered by the mouth at 3.10 p.m.

The animal became drowsy at 3.17; pupils small at 3.20, and very much contracted at 3.24; it continued sleepy, but felt when pinched, it resisted if held up, and moved about occasionally. The pupils recovered at 3.40, and the drowsiness passed off.

Exp. 41.—A man, æt. 31, in perfect health.

March 20th, at 11.35 a.m.—Two hours after breakfast  $\frac{1}{8}$  of a grain of the acetate of morphine, dissolved in m℥ of water, was injected into the cellular tissue of the left arm. Pulse 60 in the minute.

The pain of puncture was insignificant, and was not aggravated by the injection of the fluid, but was re-induced on the withdrawal of the syringe.

11.40.—Slight dizziness and faintness, which gradually increased and became severe at

11.45.—The above symptoms were then accompanied by headache, tightness of the forehead, pain at the back of the eyeballs, and slight nausea.

11.50.—Heaviness of the eyelids. P. 68. Previous symptoms increased.

11.55.—Burning heat of cheeks and face; nausea and dizziness.

12.—P. 60; dizziness increased; decided dryness of tongue; head symptoms severe; some dimness of sight; partial relief from these disagreeable symptoms was obtained by lying down.

12.7 p.m.—P. 58; headache rather less; other symptoms unaltered.

12.10.—Marked decrease in the quantity of saliva, with

general dryness of the mouth and fauces; the feeling of intoxication was so severe that there was utter incapacity for work or exertion of any kind.

12.17.—P. 54, intermittent; inclined to doze; general lassitude and disinclination to work; dryness of mouth; constriction of fauces.

12.32.—P. 53, irregular; somnolence.

1.10.—Speech thick; inclination to misuse words; pupils contracted.

1.37.—Slight itching, with general dryness of the skin.

1.55.—P. 48; is very sleepy, and slept for a short time.

3.5.—Dull and heavy, but not so overpowered with sleep.

4.20.—Enabled to go out and do some work; the more potent effects of the drug are passing off. P. irregular.

5.5.—Head a little lighter. This state continued until

11.30 p.m.—P. 50; headache; heavy eyelids; cheeks flushed; went to bed; heavy sleep disturbed by dreams, and disinclination to rise on the following morning.

March 21st.—Urine diminished in quantity; secretion of bile in the stools less than natural; perspiration diminished; appetite bad. The pulse returned to 60, at about 9.30 a.m. The dryness of the mouth lasted until 1.30 p.m.; that of the skin until about 5.30. The headache remained, though in a minor degree, until the evening of the second day, thirty-six hours after the injection; and the nausea was not removed until March 22nd, forty-six hours after the operation.

A slight soreness remained at the locality of the puncture for two days.

Exp. 42.—The same individual on March 27th, at 11.44 a.m., took  $\frac{1}{8}$  grain of acetate of morphine in  $\pi j$  of water on the tongue. P. 64.

11.54.—Slight nausea. P. 64.

12.12 p.m.—P. 64; nausea increased, and accompanied by some tightness of the forehead.

12.42.—Pain across the forehead, and nausea. P. 56; symptoms very slight, and no disinclination to work.

1.11.—Dryness of the mouth and tongue, not so marked as on the previous occasion.

1.42.—The dizziness began to pass off. P. 54. Pup not observed to be small.

1.57.—The nausea was giving place to a feeling of hunger; the headache remained, but there was less dizziness. P. 5

6.30.—P. 60: no nausea or dizziness; secretion of saliva restored; no alteration observed in the quantity of urine, bile, or perspiration; only slight heaviness existed across the brows: this had quite passed off, and no symptoms remained at 11 p.m.

EXP. 13.—A man, æt. 23, complaining only of slight pains in the soles of the feet, otherwise in good health was injected in the right arm with  $\frac{1}{4}$  of a grain of the acetate of morphine in solution, at 11.40 a.m. R. 24. P. 72 Temp. 97.9.

11.46.—P. 72. Pain across the forehead.

12.3 p.m.—R. 22. P. 72. Temp. 97.3. Dryness of throat.

12.30.—R. 22. P. 68. Temp. 97.9. Parched and flushed; intense headache, which obliged him to go to bed and lasted until sleep at 9 p.m.

EXP. 13 *bis*.—On the third day after, at 11.43 a.m., some of the same solution containing  $\frac{1}{4}$  of a grain was dropped on the tongue. P. 78. Temp. 97.8. R. 20.

11.50.—Pain across the forehead, but not so severe as before.

12.15 p.m.—P. 74. R. 20. Temp. 97.8. Nausea.

12.20.—Sleepy; all symptoms passed off at 3 p.m.

EXP. 44.—26th October.—A man, æt. 50, suffering from slight pain in the knee;  $\frac{1}{4}$  grain of the acetate of morphine was administered by the mouth in  $\frac{1}{2}$  glass of water.

2.30 p.m.—P. 84. Temp. 98. R. 24.

6.—P. 80. Temp. 97.8. R. 24. No symptoms.



Exp. 45.—October 27th, 2.30 p.m.— $\frac{1}{4}$  grain of the acetate of morphine was thrown into the rectum in  $\mathfrak{z}$ iss of water.

P. 84. Temp. 97.6. R. 24.

6.—P. 70. Temp. 96.6. R. 21. No other symptoms.

Exp. 46.—November 6th, 6.7 p.m.— $\frac{1}{4}$  grain of the acetate of morphine was injected in  $\mathfrak{m}$ iss of fluid into the arm. P. 84. R. 23. Temp. 96.2.

6.35.—P. 88. R. 20. Temp. 95.8.

7.30.—Intense itching of the skin, lasting an hour, followed by profuse sweating; drowsiness; the pulse gradually fell.

7th, 11 a.m.—P. 68. R. 20. Temp. 96.2.

Exp. 47.—November 3rd.—A man,  $\mathfrak{a}$ t. 22, suffering from slight lumbago;  $\frac{1}{4}$  grain of the acetate of morphine was injected in solution into the rectum. P. 84. R. 24. Temp. 98.

2 p.m.—P. 72. R. 24. Temp. 98.3. Itching of the head was the only symptom.

Exp. 48.—November 4th.— $\frac{1}{3}$  grain of the same in  $\mathfrak{z}$ j of water was introduced into the rectum. No effect.

Exp. 49.—November 6th.— $\frac{1}{4}$  grain in  $\mathfrak{m}$ iss of fluid was injected into the left arm.

6 p.m.—P. 96. R. 24. Temp. 98.9.

6.20.—P. 92. R. 20. Temp. 98.6. Flushes, dryness of the throat, and profuse sweating.

November 7th, 11.45 a.m.—P. 84. R. 16. Temp. 98.

5.45.—P. 100. R. 24. Temp. 99.8.

Exp. 50.—November 9th, 6.10 p.m.— $\frac{1}{3}$  grain of morphine was given by the mouth, in  $\mathfrak{z}$ ij of water. P. 88. R. 20.

6.17.—P. 86. R. 20. Temp. 98.2.

6.25.—P. 80. R. 20. Temp. 98.1. Drowsiness slight.

6.31.—P. 80. R. 20. Temp. 98.1.

No other effect observed.

Exp. 51.—November 2nd.—A man, æt. 45, in fair health;  $\frac{1}{2}$  grain of acetate of morphine in  $\mathfrak{zj}$  of water was injected into the bowel.

11 a.m.—P. 88. R. 24. Temp. 97·8.

1.30 p.m.—Sleep lasting for one hour.

2.30.—P. 80. R. 20. Intense itching of the skin.

3.—3.30.—Dozing.

5.30.—P. 72. R. 20. Temp. 97·2.

Exp. 52.—A man, æt. 60, convalescent.

11.55 a.m.— $\frac{1}{2}$  grain of morphine solution injected into the right arm. P. 66. R. 20. Temp. 96·2.

12.6 p.m.—P. 67. R. 18. Slight pain across the eyes, with flushings.

12.16.—P. 66. R. 16. Temp. 96·4.

1 p.m.—Felt as if intoxicated; was obliged to lie down, and went to sleep.

6.20.—Felt giddy; suffered from itching of the skin.

On the following day at 11 a.m. P. 66. Temp. 96. Still felt slightly giddy.

### *Quinine.*

Exp. 53.—A rather nervous girl, æt. 7 years, was the subject of experiment. The solution of quinine used, contained gr. v of the disulphate dissolved by a little sulphuric acid in  $\mathfrak{zj}$  of water.

January 9th, 11 a.m.—Temp. 99·2° (Fahr.). P. 120. Three grains of quinine were taken by the mouth.

12.0.—Temp. 99·2. P. 120.

12.20 p.m.—Temp. 99. P. 120.

2.30.—Temp. 98·6. P. 120.

Exp. 54.—Jan. 10th, 5.30 p.m.—Temp. 98·2. P. 128.

5.45.—Three grains of quinine were administered by the rectum.

Exp. 55.—Jan. 16th, 10.45 a.m.—Temp. 99.2. P. 120.  
 10.55.—Three grains of quinine were given by injection under the skin of the arm.  
 11.30.—Temp. 100.2. P. 140.  
 12.45 p.m.—Temp. 99.4. P. 132.  
 1.45.—Temp. 99. P. 120.  
 6.30.—Temp. 99. P. 120.  
 7.30.—Temp. 98.2. P. 120.

Exp. 56.—A boy, æt. 8 years, in apparent health, affected with scabies. From December 2nd to 4th the temperature in the axilla was taken ten times, and was found to range from 95.0 to 99.2. Pulse taken as frequently, 90.10.

Dec. 4th, 11 a.m.—Temp. 98. P. 95.  
 11.30.—Three grains of quinine thrown into the rectum.  
 12.10 p.m.—Temp. 99.5. P. 108.  
 12.30.—Temp. 99.5.  
 1.0.—Temp. 99.5. P. 108, rather full.  
 6 p.m.—Temp. 98.2. P. 88.

Exp. 57.—December 15th, 12 noon.—Temp. 97.5. Three grains were injected into the arm. The operation occupied nine minutes.

12.13 p.m.—Temp. 97.8.  
 12.16.—Temp. 98. Frontal headache. P. 92.  
 12.26.—Temp. 98.6. Headache. P. 100.  
 12.35.—Temp. 98.6. Headache. P. 100.  
 12.55.—Temp. 99. P. 112.  
 1.30.—Temp. 99.4. P. 104.  
 2.0.—Temp. 99.6. P. 100.  
 2.30.—Temp. 99.6. P. 100.  
 2.45.—Temp. 99.6. P. 100.  
 3.0.—Temp. 100. P. 110.  
 3.45.—Temp. 100.2. P. 120.  
 4.45.—Temp. 99. P. 98. Dinner; no appetite  
 5.30.—Temp. 98.3. P. 106.  
 December 16th, 12 noon.—Temp. 98.2. P. 88.  
 3.45 p.m.—Temp. 98.4. P. 92.

5.30.—Temp. 97·8. P. 88. Natural condition.

Exp. 58.—December 22nd, 12 noon.—Temp. 98. 1  
Three grains were given by the mouth.

1 p.m.—Temp. 99·4. P. 96.

2.0.—Temp. 99. P. 96.

3.15.—Temp. 98·2. P. 92. No headache, nor were  
other symptoms observed.

Exp. 59.—A woman, æt. 19, with slight disease o  
hip-joint.

November 2nd.—Three grains of quinine were inj  
into the outer part of the thigh of the affected  
Immediately afterwards there was pain in the knee, a  
sensation of coldness was experienced in the hip.

Exp. 60.—November 8th, 2.15 p.m.—Five grain  
quinine were injected into the outside of the thigh. P

2.40.—P. 112. Slight headache; weight across  
forehead; some giddiness.

3.20.—P. 120. Giddiness less; headache gone.

4.0.—P. 108. Face flushed; giddiness increased.

7.0.—P. 100. Less giddiness; slight headache.  
headache continued all the night, and lasted until 1  
on the 9th. No local effects.

Exp. 61.—A few days after, the same dose was give  
the rectum, the quinine being mixed with cocoa-nut bu  
No effects were observed.

Exp. 62.—A man in good health, admitted into  
hospital to be fitted with an apparatus for former rese  
of the wrist, was injected in the cellular tissue of the a  
arm with gr. v of quinine dissolved in ℥j of water  
acid. The injection was begun at 2.6 p.m., on the 14  
February, and took seventeen minutes. Before the inj  
Temp. 97·4. P. 80.

2.45 p.m.—Temp. 98. P. 88. No pain in the head.

February 15th.—He complains of stiffness in the arm, which is somewhat swollen and red. Temp. 98. P. 80.

A severe rigor occurred in the course of the evening; and next day the swelling and redness had much increased. The inflammation subsided considerably after the application of a poultice, but recurred next day owing to some slight exertion, and an abscess formed, which was opened on the 22nd of February.

### *Calabar Bean.*

An alcoholic extract was dissolved in glycerine (Bell's preparation) so that  $m_j = \text{grs. iv}$  of the powdered bean.

EXP. 63.—A small black rabbit, two months old, was injected under the skin of the loins with  $m_j$  of the above solution diluted with  $m_j$  of distilled water.

1h. 19' 15". Pupils large.

22'. Drags hind legs, 2' 45".

26' 15". Trembling and vomiting, 7'. Respiration panting.

27' 45". Trembling much, 8' 30".

28' 45". Staggering, 9' 30".

31' 15". Drags left hind leg, 11'.

33' 45". Incomplete paralysis of both hind legs, 13' 30". Pupils still widely dilated.

45'. Very lively; apparently quite recovered in 24' 45".

$m_j$  of the solution =  $\text{iv grs.}$  of the powdered bean, was injected subcutaneously, and produced incomplete paralysis.

First symptoms in 2' 45"—recovery in 24' 45".

EXP. 64.—A small black rabbit, two months old, was injected with three drops of the same solution mixed with three minims of water under the skin of the loins.

1h. 41'. Pupils dilated.

46' 30". 5' 30". Staggered.

7'. Trembling, and twitching of limbs.

7 55". Incomplete paralysis of fore legs.

8 20". Sprawling; fore and hind legs partially paralysed.

9 15". Falls upon its side.

9 45". Cannot rise.

10'. Has risen by a strong effort.

11'. Fallen again; hind legs extended and stiff.

12 15". Fore legs extended motionless; hind legs alternately flexed and extended.

12 45". Pupils contracted to half their former size.

13 20". Lying on the side.

14 30". No reflex movements could be excited.

15'. Pupils smaller; convulsive movements of limbs.

15 40". Respiration continues, though the limbs are quite paralysed.

18 45". Fore legs moved.

22'. Respiration stertorous; mucus in nares.

33 22". Convulsions.

34'. Dead; pupils dilated.

35'. Pupils contracted; subsultus.

The chest was immediately opened; lungs bloodless, collapsing on exposure; heart full on both sides, but the right most so; it continued to beat for several seconds.

mij = 12 grains of the powdered bean were injected subcutaneously, causing paralysis; death occurred in thirty-five minutes.

Exp. 65.—The same black rabbit which was used for Exp. 63; since then it has eaten well, and is quite lively.

Three minims of the solution in 5ij of water were injected through a No. 1 flexible catheter into the stomach 3.19 p.m. Pupils dilated.

3'. Respiration panting; back arched; coat staring.

5 30". Left hind leg incompletely paralysed; slight general spasm; pupils larger.

6'. Fore legs extended, motionless, relaxed; hind legs alternately flexed and extended, and, during extension, paralysed.

7'. Fore legs extended at right angles to the body.

7' 30". Respiration ceased; pupils dilated widely, then contracted and remained so.

8'. Dead; slight subsultus.

Chest opened; condition of lungs and heart identical with those in Exp. 64.

mij = 12 grains of the bean were given by the stomach, causing paralysis and death in eight minutes; the heart continued to beat after respiration had ceased; pupils contracted.

Exp. 66.—A full-grown doe rabbit; three minims of the same preparation in 3j of water were injected into the rectum at 4 h. 29 m. 30 sec. (about  $\frac{1}{3}$  was voided immediately).

Pupils dilated.

2' 15". Voided urine.

8' 30". Defecated.

No other symptoms were observed.

mij = 12 grains were administered by the rectum; no symptoms produced.

Exp. 67.—The same rabbit which was used for the last experiment. After an interval of several hours, it received mij of the solution in 3ss of water by the mouth, but no toxic effects were perceived.

mij = 12 grains were taken by the mouth. No result.

Exp. 68.—A full-grown rabbit (black buck); mij in 3ss of water were placed in the rectum.

7' 25". Voided urine.

mij = 12 grains given by the rectum produced no effect.

Exp. 69.—Into a full-grown sandy rabbit, mij mixed with an equal quantity of water were subcutaneously injected.

Pupils dilated.

9'. Strong tremor; respiration panting; paralysis of limbs.

10' 5". Fell; extended limbs, and rose with difficulty.

11' 25". Coat staring; cried faintly; staggered.

11' 45". Respiration quicker.

- 47' 30". 4'. Grinding teeth.
- 48'. 4' 30". Coat staring; panniculus quivering.
- 51' 30". 8'. Voided several pellets.
- 53'. 9' 30". Grinding teeth.
- 54'. 10' 30". Back arched; moves with very short steps.
- 54' 30". 11'. A rhythmic swaying to and fro of the body, synchronous with respiration.
- 57'. 13' 30". Rushes wildly about, striking against things in his way.
- 57' 30". 14'. A loose motion.
- 58' 30". 15'. Fell convulsed, but struggled on his legs again.
- 1h. 16' 30". Fell; hind leg extended; could not rise.
- 1h. 1'. 17' 30". Salivation; respiration rapid.
- 1h. 2' 30". 19'. A loose stool.
- 1h. 3'. 19' 30". Rose on fore legs; could not stand; cried slightly.
- 1h. 5'. 21' 30". Extended; prostrate; salivation very profuse.
- 1h. 9' 30". 26'.  $\text{miv}$  with  $\text{mij}$  of water were injected under the skin of the shoulder; the animal was lying at full length, with legs extended and head raised.
- 1h. 19' 30". 36'. 10'. Respiration slower; struggled as if to rise, but failed.
- 1h. 21' 30". 38'. 12'. No reflex movements excitable by pinching hind legs; respirations 100.
- 1h. 22' 30". 39'. 13'. Head drooping.
- 1h. 26' 30". 43'. 17'. Cried slightly.
- 1h. 35' 30". 52'. 26'.  $\text{mvij}$  more of the solution were injected under the skin of the thigh; respiration 90; pupils dilated.
- 1h. 37' 30". 54'. 28'. 2'. Tried to rise on his forelegs.
- 1h. 40' 30". 57'. 31'. 2' 30". Respiration 84.
- 1h. 43' 30". 1h. 34'. 5' 30". Voluntary movements (?) of hind legs.
- 2h. 30". 1h. 17". 51'. 22' 30". Respiration 92.
- 2h. 2' 30". 1h. 19'. 53'. 24' 30". Effort at progression; clonic spasm of thighs.



140 grains of the bean, were taken in 1 hour, 35 min. 30 sec. Paralysis; profuse salivation; purging; recovery.

Exp. 71.—The same rabbit used for the last experiment four days ago. His paralysis has quite disappeared, but he looks poorly; coat rough; is purged, and does not eat.

Half a drachm of solution with 3ss of water was injected into the rectum.

6' 45". Respiration panting.

7'. A loose stool.

Thinking that most of the solution had been voided, ℥xv of solution with 3ss of water were injected into the stomach.

13'. 2' 30". Extended prostrate.

14' 30". 4'. Fallen on side.

15' 45". 5' 15". Convulsed; pupils dilated.

17' 30". 7'. Inspiration at long intervals; insensible; subsultus of fore paw.

18'. 7' 30". Death; pupils contracted.

Lungs pale and collapsed on exposure; heart pulsating feebly, its right rather fuller than its left side. Stomach, cardiac portion, coated with a thick white pellicle (lymph?); pyloric portion purplish from congestion; distended with thick mucus and wind.

3ss of the solution was given by the rectum, and ℥xv by the mouth, together = 180 grains of the bean. Paralysis; death in eighteen minutes.

Exp. 72.—A full-grown grey buck rabbit. 3ss of the solution with 3ss of water was given by the mouth, but nearly half was lost, as the fluid returned in consequence of the tube being too short.

10' 45". Rather unsteady in its gait.

18'. Tremors.

19' 25". Hind legs extended, and with difficulty drawn up again.

27' 30". Flanks tucked in.

28'. A loose stool.

No signs of paralysis appearing,  $\pi\alpha\lambda$  in  $\pi\alpha\alpha$  of water were injected into the stomach.

30 50 . 2 50 . Grinding teeth.

30 20 . 22 20 . No further effects observable.

$\pi\alpha\lambda$  of the solution in  $\pi\alpha\alpha$  of water were again injected into the stomach.

1 2 . 20 2 . 1. Pupils contracted.

18 1 . 30 1 . 7 45 . Hind legs extended.

30 5 . 31 5 . 8 45 . Lying on side.

1h. 1 25 . 33 25 . 11 5 . Hind legs again extended.

1h. 4 10 . 36 10 . 13 45 . Rhythmical swaying to and fro, synchronous with respiration.

1h. 10 . 38 35 . 15 45 . Great trembling; loud cries.

1h. 18 . 50 . 27 40 . Extended full length: cannot rise: wavy tremors of panniculus.

1h. 19 . 51 . 28 40 . Gurgling, as of wind in belly.

1h. 27 . 5 . 30 4 . Head fallen on left side.

1h. 28 . 1h. 37 4 . Strychnine  $\gamma$ th grain in  $\pi\eta$  injected under the skin of the loins.

1h. 29 . 1h. 1 . 38 40 . 1. A slight convulsion.

1h. 30 30 . 1h. 2 30 . 40 10 . 2 30 . Slight convulsions.

1h. 31 . 1h. 3 . 40 4 . 3. Slight twitchings: dead.

Heart beating, right side fuller than left: lungs pale: stomach contained much food and wind: pyloric end congested and ecchymosed.

$\pi\alpha\alpha\alpha - \alpha\lambda - \alpha\lambda = \pi\alpha\alpha$  of the solution = 440 grains of beer were injected into the stomach: death in 1h. 31 .

The rabbit was in articulo mortis before the strychnine was injected.

### *Conia.*

The drug was obtained from Morson, of Southampton Row, in two solutions—the first contained one grain of the alkaloid in  $\gamma\eta$  of water, to which had been added a little dilute sulphuric acid, so to make it as nearly neutral as possible; the second contained four grains in  $\gamma\eta$ .

The solution was clear; faintly acid; pale brown in colour;

pungently odorous, like oil of tobacco; taste, at first, cool, as is the case with the volatile oils, soon becoming intensely acrid, producing tingling of the part touched, and causing ultimately numbness of long duration.

*Skin.*

EXP. 73.—A young rabbit, weighing 5 lbs., was injected at 3.34 p.m. with gr.  $1\frac{1}{2}$  of conia in  $\text{ʒiij}$  of fluid under the skin of the back.

In 26' it became quiet.

31' it was attacked with general tremors and arching of the back (emprosthotonos); breathing spasmodic and slow.

34', general spasms; pulse slow, 88.

36', breathing ceased; pulse accelerated; the heart continued to beat for three minutes after the respiration had stopped.

39', death complete. Pupils contracted strongly after death.

Rigor mortis fully established in  $1\frac{1}{2}$  hours.

A smaller dose was next tried.

EXP. 74.—A young rabbit (X), weight  $4\frac{3}{4}$  lbs., was injected with gr.  $\frac{1}{2}$  in solution under the skin of the back. Became quiet in seventeen minutes.

In 19', moved clumsily.

20', respiration more rapid and irregular.

22', moved about clumsily.

30', began to recover. No further symptoms observed. Was quite well on the following day.

EXP. 75.—A young rabbit, weight about  $4\frac{1}{4}$  lbs. (male), was injected with gr.  $\frac{1}{2}$  in 15 m of fluid under the skin of the back.

In 17', fell sprawling, with hind legs extended; after several successive falls, the breathing increased in rapidity; great hyperæsthesia; hind legs drawn up spasmodically on being touched; no expression of pain.

530. Temp. 97.8. P. 88. Natural condition.

EXR. 58. December 22nd, 12 noon.—Temp. 98. P. Three grains were given by the mouth.

1 p.m. Temp. 99.4. P. 96.

2.0 Temp. 99. P. 96.

3.45 Temp. 98.2. P. 92. No headache, nor were other symptoms observed.

EXR. 59. A woman, æt. 19, with slight disease of hip-joint.

November 2nd. Three grains of quinine were injected into the outer part of the thigh of the affected hip. Immediately afterwards there was pain in the knee, and sensation of coldness was experienced in the hip.

EXR. 60. November 8th, 2.15 p.m.—Five grain quinine were injected into the outside of the thigh. P.

2.40 P. 112. Slight headache; weight across forehead; some giddiness.

3.20. P. 120. Giddiness less; headache gone.

4.0 P. 108. Face flushed; giddiness increased.

7.0 P. 110. Less giddiness; slight headache. Headache continued all the night, and lasted until 1.0 on the 9th. No local effects.

EXR. 61. A few days after, the same dose was given into the rectum, the quinine being mixed with cocoa-nut butter. No effects were observed.

EXR. 62. A man in good health, admitted into hospital to be fitted with an apparatus for former *resaca* of the wrist, was injected in the cellular tissue of the *extensor* arm with gr. v of quinine dissolved in 3j of water acid. The injection was begun at 2.6 p.m., on the 14th February, and took seventeen minutes. Before the injection Temp. 97.4. P. 80.

2.45 p.m.—Temp. 98. P. 88. No pain in the head.

February 15th.—He complains of stiffness in the arm, which is somewhat swollen and red. Temp. 98. P. 80.

A severe rigor occurred in the course of the evening; and next day the swelling and redness had much increased. The inflammation subsided considerably after the application of a poultice, but recurred next day owing to some slight exertion, and an abscess formed, which was opened on the 22nd of February.

### *Calabar Bean.*

An alcoholic extract was dissolved in glycerine (Bell's preparation) so that  $\text{mj} = \text{grs. iv}$  of the powdered bean.

EXP. 63.—A small black rabbit, two months old, was injected under the skin of the loins with  $\text{mj}$  of the above solution diluted with  $\text{mj}$  of distilled water.

1h. 19' 15". Pupils large.

22'. Drags hind legs, 2' 45".

26' 15". Trembling and vomiting, 7'. Respiration panting.

27' 45". Trembling much, 8' 30".

28' 45". Staggering, 9' 30".

31' 15". Drags left hind leg, 11'.

33' 45". Incomplete paralysis of both hind legs, 13' 30". Pupils still widely dilated.

45'. Very lively; apparently quite recovered in 24' 45".

$\text{mj}$  of the solution =  $\text{iv grs.}$  of the powdered bean, was injected subcutaneously, and produced incomplete paralysis.

First symptoms in 2' 45"—recovery in 24' 45".

EXP. 64.—A small black rabbit, two months old, was injected with three drops of the same solution mixed with three minims of water under the skin of the loins.

1h. 41'. Pupils dilated.

46' 30". 5' 30". Staggered.

7'. Trembling, and twitching of limbs.

7' 30". Respiration ceased; pupils dilated widely, then contracted and remained so.

8'. Dead; slight subsultus.

Chest opened; condition of lungs and heart identical with those in Exp. 64.

$\text{mij} = 12$  grains of the bean were given by the stomach, causing paralysis and death in eight minutes; the heart continued to beat after respiration had ceased; pupils contracted.

Exp. 66.—A full-grown doe rabbit; three minims of the same preparation in  $\text{zj}$  of water were injected into the rectum at 4 h. 29 m. 30 sec. (about  $\frac{1}{2}$  was voided immediately).

Pupils dilated.

2' 15". Voided urine.

8' 30". Defecated.

No other symptoms were observed.

$\text{mij} = 12$  grains were administered by the rectum; no symptoms produced.

Exp. 67.—The same rabbit which was used for the last experiment. After an interval of several hours, it received  $\text{mij}$  of the solution in  $\text{zss}$  of water by the mouth, but no toxic effects were perceived.

$\text{mij} = 12$  grains were taken by the mouth. No result.

Exp. 68.—A full-grown rabbit (black buck);  $\text{mij}$  in  $\text{zss}$  of water were placed in the rectum.

7' 25". Voided urine.

$\text{mij} = 12$  grains given by the rectum produced no effect.

Exp. 69.—Into a full-grown sandy rabbit,  $\text{mij}$  mixed with an equal quantity of water were subcutaneously injected.

Pupils dilated.

9'. Strong tremor; respiration panting; paralysis of limbs.

10' 5". Fell; extended limbs, and rose with difficulty.

11' 25". Coat staring; cried faintly; staggered.

11' 45". Respiration quicker.

- 47' 30". 4'. Grinding teeth.
- 48'. 4' 30". Coat staring; panniculus quivering.
- 51' 30". 8'. Voided several pellets.
- 53'. 9' 30". Grinding teeth.
- 54'. 10' 30". Back arched; moves with very short steps.
- 54' 30". 11'. A rhythmic swaying to and fro of the body, synchronous with respiration.
- 57'. 13' 30". Rushes wildly about, striking against things in his way.
- 57' 30". 14'. A loose motion.
- 58' 30". 15'. Fell convulsed, but struggled on his legs again.
- 1h. 16' 30". Fell; hind leg extended; could not rise.
- 1h. 1'. 17' 30". Salivation; respiration rapid.
- 1h. 2' 30". 19'. A loose stool.
- 1h. 3'. 19' 30". Rose on fore legs; could not stand; cried slightly.
- 1h. 5'. 21' 30". Extended; prostrate; salivation very profuse.
- 1h. 9' 30". 26'.  $\text{miv}$  with  $\text{mij}$  of water were injected under the skin of the shoulder; the animal was lying at full length, with legs extended and head raised.
- 1h. 19' 30". 36'. 10'. Respiration slower; struggled as if to rise, but failed.
- 1h. 21' 30". 38'. 12'. No reflex movements excitable by pinching hind legs; respirations 100.
- 1h. 22' 30". 39'. 13'. Head drooping.
- 1h. 26' 30". 43'. 17'. Cried slightly.
- 1h. 35' 30". 52'. 26'.  $\text{mvij}$  more of the solution were injected under the skin of the thigh; respiration 90; pupils dilated.
- 1h. 37' 30". 54'. 28'. 2'. Tried to rise on his forelegs.
- 1h. 40' 30". 57'. 31'. 2' 30". Respiration 84.
- 1h. 43' 30". 1h. 34'. 5' 30". Voluntary movements (?) of hind legs.
- 2h. 30". 1h. 17". 51'. 22' 30". Respiration 92.
- 2h. 2' 30". 1h. 19'. 53'. 24' 30". Effort at progression; clonic spasm of thighs.

140 grains of the bean, were taken in 1 hour, 35 min. 30 sec. Paralysis; profuse salivation; purging; recovery.

Exp. 71.—The same rabbit used for the last experiment four days ago. His paralysis has quite disappeared, but he looks poorly; coat rough; is purged, and does not eat.

Half a drachm of solution with  $\text{zss}$  of water was injected into the rectum.

6' 45". Respiration panting.

7'. A loose stool.

Thinking that most of the solution had been voided,  $\text{mxxv}$  of solution with  $\text{zss}$  of water were injected into the stomach.

13'. 2' 30". Extended prostrate.

14' 30". 4'. Fallen on side.

15' 45". 5' 15". Convulsed; pupils dilated.

17' 30". 7'. Inspiration at long intervals; insensible; subsultus of fore paw.

18'. 7' 30". Death; pupils contracted.

Lungs pale and collapsed on exposure; heart pulsating feebly, its right rather fuller than its left side. Stomach, cardiac portion, coated with a thick white pellicle (lymph?); pyloric portion purplish from congestion; distended with thick mucus and wind.

$\text{zss}$  of the solution was given by the rectum, and  $\text{mxxv}$  by the mouth, together = 180 grains of the bean. Paralysis; death in eighteen minutes.

Exp. 72.—A full-grown grey buck rabbit.  $\text{zss}$  of the solution with  $\text{zss}$  of water was given by the mouth, but nearly half was lost, as the fluid returned in consequence of the tube being too short.

10' 45". Rather unsteady in its gait.

18'. Tremors.

19' 25". Hind legs extended, and with difficulty drawn up again.

27' 30". Flanks tucked in.

28'. A loose stool.



pungently odorous, like oil of tobacco ; taste, at first, cool, as is the case with the volatile oils, soon becoming intensely acrid, producing tingling of the part touched, and causing ultimately numbness of long duration.

*Skin.*

EXP. 73.—A young rabbit, weighing 5 lbs., was injected at 3.34 p.m. with gr.  $1\frac{1}{2}$  of conia in  $\text{ʒiij}$  of fluid under the skin of the back.

In 26' it became quiet.

31' it was attacked with general tremors and arching of the back (emprosthotonos) ; breathing spasmodic and slow.

34', general spasms ; pulse slow, 88.

36', breathing ceased ; pulse accelerated ; the heart continued to beat for three minutes after the respiration had stopped.

39', death complete. Pupils contracted strongly after death.

Rigor mortis fully established in  $1\frac{1}{2}$  hours.

A smaller dose was next tried.

EXP. 74.—A young rabbit (X), weight  $4\frac{1}{2}$  lbs., was injected with gr.  $\frac{1}{2}$  in solution under the skin of the back. Became quiet in seventeen minutes.

In 19', moved clumsily.

20', respiration more rapid and irregular.

22', moved about clumsily.

30', began to recover. No further symptoms observed. Was quite well on the following day.

EXP. 75.—A young rabbit, weight about  $4\frac{1}{2}$  lbs. (male), was injected with gr.  $\frac{1}{2}$  in 15  $\text{m}$  of fluid under the skin of the back.

In 17', fell sprawling, with hind legs extended ; after several successive falls, the breathing increased in rapidity ; great hyperæsthesia ; hind legs drawn up spasmodically on being touched ; no expression of pain.

A solution of cold starch paste and chloride of lime was used for detecting the iodide, and the urine was allowed to trickle into a number of test tubes containing the solution.

The man was dieted; and the urine throughout the experiments flowed from the ureters at a uniform rate.

EXP. 125.—Breakfast at 7 a.m. At 2 p.m. (7 hours' fasting) gr. iv of the iodide of potassium in  $\text{m}\times$  of water were placed on the tongue. Distinct reaction was observed in  $14\frac{1}{4}$  minutes.

EXP. 126.—Dinner at 12.30. At 2 p.m. one grain of the iodide in  $\text{m}\text{iiss}$  of water was injected into the right arm. The reaction was obtained in  $20\frac{1}{4}$  minutes.

The injection gave rise to much pain and smarting; the surrounding parts were very red, and the irritation was so severe as to keep the patient awake until 2 a.m. A small ulcer resulted, which remained open for several days.

EXP. 127.—Dinner 12.30. At 2 p.m. gr. iv of the iodide in  $\text{m}\times$  of water were dropped on the tongue. Reaction in  $14\frac{1}{2}$  minutes.

EXP. 128.—Breakfast at 7.30. At 11.30 a.m. the same dose was given by the mouth. Reaction in 14—15 minutes.

EXP. 129.—At 11.30 a.m., 4 hours after breakfast, gr. iv in  $\text{m}\times$  of water were injected into the rectum. The bowels had acted the previous night. No reaction was obtained after waiting 3 hours.

EXP. 130.—At 11.30 a.m., the bowels having been opened before 7 a.m., the same dose was injected into the rectum. No effect.

EXP. 131.—At 11.15 a.m., the bowels having acted at 9 a.m., gr. iv in  $\text{zss}$  of water were injected into the rectum. Reaction was obtained in 23 minutes.

## CASES TREATED BY SUBCUTANEOUS INJECTION.

*Experiments with Aconitine on Man.*

CASE 1.—Fred. S—, æt. 28, had suffered from sciatica for some time. A tender spot was found over the last lumbar vertebra; pain, however, was referred to the sciatic notch.  $\frac{mij}{10}$  of a solution of aconitine containing  $\frac{1}{320}$ th of a grain was injected subcutaneously. Considerable smarting resulted, which lasted for seven hours. No alleviation of the neuralgic pain was experienced.

CASE 2.— $\frac{mij}{10}$  of the same solution =  $\frac{1}{256}$ th of a grain were subcutaneously injected; the smarting after injection was very considerable. No alleviation of pain resulted.

*Atropine injections.*

CASE 3.—Luke S—, æt. 56, suffered from excruciating pains in the right knee, depending upon malignant disease.

September 29th.—Was injected with  $\frac{1}{4}$  grain of acetate of morphine. No sleep.

October 4th.—The injection was repeated.

6th.— $\frac{1}{4}$  grain was injected, which relieved the pain so that he slept during that night and the following day. It returned, however, and tincture of aconite was administered, but without benefit. The patient was unable to move his right leg from the pain.

30th.— $\frac{1}{32}$ th grain of atropine was injected, but without relief. Pupils natural; dry mouth and much thirst; no sleep.

31st.— $\frac{1}{32}$ th grain of atropine was injected at 7 p.m. At 8.45 p.m. he became delirious, crawled about his bed, talked at random, and poked the other patients about with a stick, but was not violent; he was able to get out of bed without difficulty; there was no pain next morning, but he had not slept; pupils natural.

November 1st.—Pain relieved; he is still slightly delirious.

3rd.—Patient occasionally rambles in talking; the pain returned on the 7th.

7th.— $\frac{1}{4}$ th of a grain of atropine was injected, which gave relief; it produced no delirium, but dryness of mouth.

There was no permanent alleviation.

CASE 4.—James E—, æt. 57, severe sciatica of eight weeks' date. Was injected with (as much as) 1 grain of the acetate of morphine without relief. Subsequently he was injected with atropine, the dose being gradually increased until the  $\frac{1}{10}$ th grain was given.

April 2.— $\frac{1}{10}$ th grain injected; it produced delirium and great dryness of throat.

3rd.— $\frac{1}{10}$ th grain repeated. The patient stated that the repetition had far less effect than the previous injection; no dryness of the throat nor delirium were produced.

The pain was so far relieved that he could walk.

During the time that this patient was under the influence of atropine, he experienced great difficulty in passing water.

CASE 5.—Wm. C—, æt. 27, suffering from sciatica of four months' duration. Was injected with  $\frac{1}{4}$ th grain of atropine.

6.11 p.m.—P. 86.

6.30.—P. 130; he was dizzy, nauseated and flushed; the pain was numbed; much dryness of mouth and fauces.

9.45.—P. 92; pupils natural; giddiness and nausea passing off.

#### *Injections with Morphine.*

CASE 6.—Kenrith M—, æt. 26, sciatica of the right leg for two months.

October 25th.— $\frac{1}{4}$  grain of the acetate was given by the mouth (the bowels having been open at 8 a.m.).

At 2.30 p.m.—P. 80. R. 28. Temp. 97.6.

At 6 p.m.—P. 68. R. 20. Temp. 98.4. Pain as before.

On the 27th, at 2.30 p.m.— $\frac{1}{4}$  grain was injected into the rectum. P. 76. R. 24. Temp. 97.7.

At 6 p.m.—P. 62. R. 20. Temp. 97·6. No symptoms of the presence of the drug, but the pain relieved slightly.

October 30th.—In the evening P. 76. R. 24. Temp. 97·6. He was injected subcutaneously at 10 p.m. with  $\frac{1}{4}$  grain. Was prevented from sleeping by considerable itching of the skin, which lasted for seven hours. The pain was numbed and relieved.

31st.—In the evening at 6 p.m. P. 58. R. 22. Temp. 97·6.

CASE 7.—John G—, æt. 35, sciatica of left leg for two months.

Nov. 9th, 5.40 p.m.—Was injected subcutaneously with  $\frac{1}{4}$  grain of the acetate. P. 88. R. 27. Temp. 99.

5.45.—P. 94. R. 26. Temp. 99·3.

5.50.—P. 94. R. 23. Temp. 99·1. Giddiness and headache.

5.55.—P. 92. R. 21. Temp. 98·6. Less giddiness.

6.10.—P. 90. R. 21. Temp. 98·6.

6.25.—P. 80. R. 21. Temp. 98·2. Pain slightly relieved.

CASE 8.—James B., æt. 26, admitted with slight sciatica of the right leg, of two weeks' duration.

26th.— $\frac{1}{4}$  grain of the acetate of morphine was taken by the mouth. No relief from pain, but sleep for two hours.

27th.— $\frac{1}{4}$  grain was given by the bowel; pain less, but not entirely removed, and it returned in fourteen hours; eight hours' sleep was, however, obtained.

30th.— $\frac{1}{4}$  grain was injected into the arm; the pain was relieved, not to return.

CASE 9.—James E—, æt. 57. Severe neuralgia of two months' duration, resulting from disease of the hip.

$\frac{1}{4}$  grain,  $\frac{2}{3}$  grain, and 1 grain, were successively injected subcutaneously, but produced no relief.

CASE 10.—George A—, æt. 53, sciatica of twelve months' date. This man took  $\frac{1}{4}$  grain by the mouth without benefit.  $\frac{1}{4}$  grain injected subcutaneously decidedly relieved the pain.



CASE 11.—Henry H—, æt. 50. Suffering for five days with pain in the right hip (of rheumatic character).

$\frac{1}{4}$  grain by the mouth and by the rectum failed to relieve ;  
 $\frac{1}{4}$  grain injected subcutaneously removed the pain.

CASE 12.—A man with lumbago of three weeks' duration.  $\frac{1}{4}$  grain given by the bowel and  $\frac{1}{4}$  grain by the mouth produced no effect on the pain ; but  $\frac{1}{4}$  grain introduced subcutaneously removed it entirely for sixteen hours. The pain subsequently yielded to other remedies.

CASE 13.—A groom was admitted with symptoms of incipient delirium tremens, accompanied by rheumatismal swelling of the joints. He had not slept for four nights previous to admission ; an aperient was administered, and on the following night  $\frac{3}{4}$  of a grain of the acetate of morphine, in  $\mathfrak{z}$ ij of water, was administered by the mouth. Very short, fitful, and restless dozing resulted ; the symptoms were not relieved.

The following night  $\frac{1}{4}$  grain of the acetate of morphine was injected into the arm. Sleep ensued in an hour and a half, and lasted during the night ; the symptoms were much relieved, the sleep having been not only more profound, but longer.

CASE 14.—A tailor's clerk, who, after giving up drinking for some time, had returned to his old habits, and was suffering from severe delirium tremens. Had taken  $\frac{1}{4}$  grain doses of acetate of morphine at night, and  $\mathfrak{mxx}$  of liq. morphinæ hydrochloratis three times a day, for two days, until the pupils were contracted, and yet no sleep was procured. The pupils were allowed to recover their natural state and  $\frac{3}{4}$  of a grain was then injected into the arm ; the patient went to sleep in  $\frac{3}{4}$  of an hour, and no further opiate was given.

CASE 15.—An auctioneer's porter, after an epileptic fit, was attacked with symptoms of delirium tremens. He was extremely violent, and it was found necessary to put on the strait-waistcoat. He was ordered  $\mathfrak{3j}$  of antimonial wine with

℥xx of liq. opii sedat. = 2 grs. of opium, every four hours; of this he took four doses, = 8 grains, and though no sleep was procured, the pupils were contracted to a pin's point. He was then given ʒj of Tinct. digitalis every four hours, but this produced no good effect. The pupils having returned to their natural condition, on the fifth night of sleeplessness he was injected with  $\frac{1}{4}$  grain of acetate of morphine, which procured two hours' sleep; but he woke in a more violent state than before, and throughout the following day he remained in a most critical condition.

On the sixth night 1 grain of morphine was injected, and he fell asleep in an hour (7.30). The drug affected his breathing so much that it was thought for a short time during the night that each breath would be his last. He, however, recovered, and the next morning he woke in his right mind, and remained quiet and sensible, sleeping well the following nights without further doses of opium.

CASE 16.—A man was admitted on November 16th with delirium tremens of two days' date (this was his second attack). He was so violent as to require restraint; his pulse was weak, 120; skin cold and perspiring.

12 noon.—He was ordered ℥xxx of laudanum. Two hours afterwards (2 p.m.) he was subcutaneously injected with ℥xij of liquor opii sedativus. Besides this he took, by the mouth, ℥xv of liq. opii sed. every four hours.

9 p.m.—The bowels were opened with senna. He was again injected with ℥xij of the liquor, the symptoms not having been relieved.

12.—A repetition of ℥xij. Sleep restless, but he dozed for some time.

17th.—Very excitable, but better. No sleep during the day.

18th.—No visible improvement. At night ℥x of the liquor opii were injected by the skin, but without producing any effect. ℥xxx were also given by the mouth.

19th.—Very fidgety. Ordered liq. opii ℥xv, 6tis horis. ℥xij were subcutaneously injected at night. He was very violent.

9th.— $\frac{1}{3}$  of a grain of acetate of morphine was injected the following night without any result.

10th.— $\frac{2}{3}$  of a grain were injected without effect; the bowels have not been moved for several days, and the tongue is much furred.

11th.—A turpentine enema was administered, and the bowels were freely opened by 5.30 p.m. At 6, gr.  $\frac{2}{3}$  of morphine were injected, and he slept well for four hours, awaking more quiet; but towards the following night (12th) he became very noisy, and at 9 p.m. one grain was injected without effect; this was followed by a second injection of 1 grain at 2 a.m. (13th), when he slept four hours, and awoke much quieter; this tranquil state continued until the 16th.

He was not injected again, but finally died worn out.

CASE 19.—A man, *æt. circ.* 45. Cancer of heart; excruciating pain in the left side and in the spine, with great restlessness and want of sleep.

Subcutaneous injections were used during one month, in doses of  $\frac{1}{4}$ ,  $\frac{1}{3}$ ,  $\frac{2}{3}$ , and at last 1 grain of acetate of morphine.

The pain began to diminish in from ten to fifteen minutes after injection, and perfect ease was experienced in about half an hour. After six hours pain and restlessness returned, and at the end of the eighth hour the effects of the injection had quite worn off.

For many days 1 grain was injected three times in the twenty-four hours at equal intervals. No nausea or stupor were produced, but ease was obtained and sleep not deeper than natural, from which he easily awoke, and after which he was always refreshed.

Injections were made in the side, in the back, and in the arms, with the same results as regards the relief of the pain and the duration of the effects.

CASE 20.—An old woman, *æt. circ.* 70, had  $\frac{1}{4}$  grain injected over the infra-orbital foramen, for severe tic in the superior maxillary division of the fifth nerve. She became very drowsy, and could with difficulty be roused during several



hours by shouting to her; as the stupor passed off she retched violently.

CASE 21.—A woman, *æt. circ.* 45, had  $\frac{1}{2}$  grain injected into the left temple, for severe neuralgia in a scar resulting from a scald.

She became very drowsy, was with great difficulty roused, and retched for some hours.

CASE 22.—A. B—, *æt.* 50, a porter, admitted into King's College Hospital, in 1859. He was suffering from severe pain in the course of the sciatic nerve, of many weeks' duration.  $\mathfrak{mxxv}$  of the liq. morph. acet. were injected subcutaneously in the region of the sciatic notch. The patient soon became drowsy, and this appeared to be deepening into coma, from which it was difficult to rouse him by shaking and shouting. He continued in this state for two or three hours, but finally recovered.

The pain ceased after the operation, but returned in a very mild and transient form a few days afterwards.

CASE 23.—A lady, *æt.* 24, had suffered for many years from severe neuralgia, which treatment had failed to relieve. At 9 p.m.  $\mathfrak{m xv}$  of a solution, =  $\frac{1}{2}$  grain of hydrochlorate of morphine, were injected into the leg. In five minutes a feeling of sinking about the heart was experienced, with inability to stand: repeated attacks of syncope followed, recurring at irregular but frequent intervals for about four hours. Whilst these lasted her countenance was livid and anxious, she was pulseless and appeared at the point of death: after they had passed off she was conscious but extremely prostrate; she could tell when the attacks were coming on by the distressing sensation over the region of the heart: there was no stupor or narcotism. Stimulants were freely employed and galvanism, but it was not until four hours had elapsed that the attacks began to diminish in frequency and severity: about 3 o'clock she fell asleep. During the next two days she had a few slighter attacks, and continued so exhausted for a fortnight as to be unable to leave her couch.

There was no return of the neuralgia for many months, although previous to the injection she had suffered from it daily.

*Injections with Quinine.*

CASE 24.—Thos. S—, æt. 28, admitted on account of pain in the situation of the right sciatic nerve.

Feb. 14th, 1.20 p.m.—Temp. 99°. P. 100. Quinæ disulph. gr. iij in ʒss of water were injected under the skin of the right buttock. The operation lasted eight minutes.

1.35.—Temp. 99°. P. 94.

1.45.—Temp. 99·8°. P. 96.

4.45.—Temp. 100·4°. P. 92.

Feb. 15th, 2 p.m.—Temp. 99·6°. P. 100. He complained of much tenderness at the part operated on, but this soon subsided.

The injection was followed by temporary relief to the pain.

CASE 25.—George O—, æt. 24, groom. Seized on the 1st of May with an attack of ague. Admitted on the 7th with all the symptoms of tertian ague (this man had lived five weeks in Park Lane and for five months previously at Romford). The rigors came on generally at 11.30 a.m. He was ordered grain ij of quinine three times a day, the bowels having just been cleared by an aperient.

On the 10th, the further addition of ℥xv of laudanum was ordered two or three hours before the fit came on.

This treatment was continued up to the 16th without any alteration in the severity of the fits.

On the 17th inst., as the cold stage was beginning, at 11.15 a.m., gr. v of quinine in ʒj of water were given by the mouth. P. 90; the bowels had been freely opened.

11.25 a.m.—P. 84. Temp. 100.

12.10 p.m.—P. 116. Temp. 102. Very cold, and shivering. 2.50.—P. 96. Temp. 103. Very hot.

The fit lasted until 6 in the evening.



18th.—Grains  $v$  in  $3j$  of water were again given by the mouth, at 10.25 a.m., as the attack was evidently commencing; at 10.35 the temperature was increased to  $100\cdot2$ . P. 99.

11.30 a.m.—Temp. 102. P. 112. Rigors.

1 p.m.—Temp. 105. P. 140. Rigors not so severe.

The fit went off at 8 o'clock; bowels were opened at 7.

No further medicine was given until the 21st, when, at 10.10 a.m., gr.  $v$  of quinine were injected into the right arm, as the symptoms were coming on. P. 72. Temp.  $97\cdot5$ .

10.35 a.m.—P. 72. Temp.  $98\cdot6$

11.0.—P. 76. Temp.  $98\cdot1$ . Slight headache.

11.15.—P. 76. Temp.  $97\cdot9$ .

11.30.—P. 76. Temp.  $97\cdot9$ .

12.0.—P. 72. Temp.  $97\cdot2$ .

No fit occurred: nor was there the slightest threatening of an attack afterwards.

### *Experiments with Podophyllin.*

CASE 1.—December 3rd, 1865.—A man, æt. 37, suffering from sciatica, but otherwise healthy. His bowels had not been moved for forty-eight hours; they generally act every morning regularly before breakfast.  $\mathfrak{mij}$  of a solution, containing  $\frac{1}{4}$  grain of podophyllin, were injected into the subcutaneous fat of the nates. It caused pain, not severe, which passed off in ten minutes. Exactly three hours afterwards his bowels were opened without previous griping; the motion was copious and solid. Next day there was still some tenderness and induration at the seat of puncture, but on the day following this had disappeared.

CASE 2.—January 15th, 1866.—A woman, with vesico-vaginal fistula, in fair health. Her bowels had not acted for five days;  $\mathfrak{mij}$  of the same solution,  $= \frac{1}{10}$  grain, were injected into the forearm. She complained of pain at the time, and for some days there was a tender swelling at the seat of puncture; no effect on the bowels was produced.

CASE 3.—December 3rd, 1866.—An hysterical woman, æt. 25, suffering from constipation of uncertain duration, probably a week.  $\text{mij}$  of the solution,  $= \frac{3}{10}$  grain, were injected into the forearm; she cried with the pain for two or three minutes. Next day there was a little tenderness and induration. No action of the bowels was obtained.

CASE 4.—A girl, æt. 8, suffering from chorea. Bowels not open for sixty hours; has suffered previously from habitual constipation.  $\text{mj}$  of the solution, with  $\text{mj}$  of water,  $= \frac{1}{10}$ th grain of podophyllin was injected into the right forearm. She cried, but in three minutes ceased to complain of pain. Bowels opened freely in twelve hours.

February 6th.— $\text{mij}$  of the solution,  $= \frac{1}{3}$  grain, with  $\text{mj}$  of water were injected. Twenty-eight hours afterwards the bowels were scantily moved. Three days after the injection there was a hard swelling on the forearm, like a large boil.

CASE 5.—A woman, æt. 58. Dispensary patient. Atonic dyspepsia; constipation; slight jaundice.

*By the stomach,*  $\text{mij}$  of undiluted solution,  $= \frac{3}{10}$  grain, in a slightly alkaline mixture three times a day, failed to produce a daily motion.

*Subcutaneously,*  $\text{miv}$  of a diluted solution,  $= \frac{1}{3}$  grain, were followed by a solid but ample stool in six hours, all other medicines being withheld for that day.

CASE 6.—A woman, æt. 40. Dispensary patient. Succession of boils on the left arm; sallow; anæmic; very costive.

*By the stomach,*  $\text{mij}$  of undiluted solution,  $= \frac{3}{10}$  grain, in a slightly alkaline carminative mixture, failed to produce a motion every day.

*Subcutaneously,*  $\text{miv}$  of a diluted solution,  $= \frac{1}{3}$  grain, produced an ample motion in six hours. After a lapse of ten days the same dose, administered in the same way, took twenty-four hours to act.

of the motion was altered and no longer pultaceous and pale, but dark in colour and without blood.

This condition has been maintained by the internal exhibition of the solution since.

CASE 11.—A man, æt. 16. Dispensary patient. Ulceration of mucous membrane of nares; œdema of face; great swelling of nose; remained very costive in spite of  $\frac{1}{4}$  grain of podophyllin in pill every night.

*By the stomach*, the effect was almost *nil*;  $\frac{1}{4}$  grain every night was ineffectual to produce motions oftener than once in two or three days.

*Subcutaneously*,  $\mathfrak{m}\text{ij}$  of the undiluted solution, =  $\frac{2}{10}$  grain, with  $\mathfrak{m}\text{j}$  of water ( $\mathfrak{m}\text{iv}$  in all), produced a solid motion in eight hours.

After the subcutaneous injections, the bowels acted daily by the use of the same means which were ineffectual before.

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